CLINICOEPIDEMIOLOGIC STUDY

OF PHYTODERMATITIS

Dissertation Submitted in

Partial fulfillment of the University regulations for

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DERMATOLOGY, VENEREOLOGY AND LEPROSY
(BRANCH XX)

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CERTIFICATE

Certified that this dissertation entitled **"CLINICOEPIDEMIOLOGIC STUDY OF PHYTODERMATITIS"** is a bonafide work done by **Dr.S.SASIREKHA,** Post graduate student of the Department of Dermatology, Venereology and Leprosy, Madras Medical College, Chennai – 3, during the academic year 2010 – 2013. This work has not previously formed the basis for the award of any degree.

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DECLARATION

I, **Dr.S.SASIREKHA** solemnly declare that this dissertation titled "**CLINICOEPIDEMIOLOGIC STUDY OF PHYTODERMATITIS**" is a bonafide work done by me at Madras Medical College during 2010-2013 under the guidance and supervision of **Prof.Dr.K. MANOHARAN, M.D.,D.D.,** Professor and Head of the Department of Dermatology, Madras Medical College,Chennai-600003.

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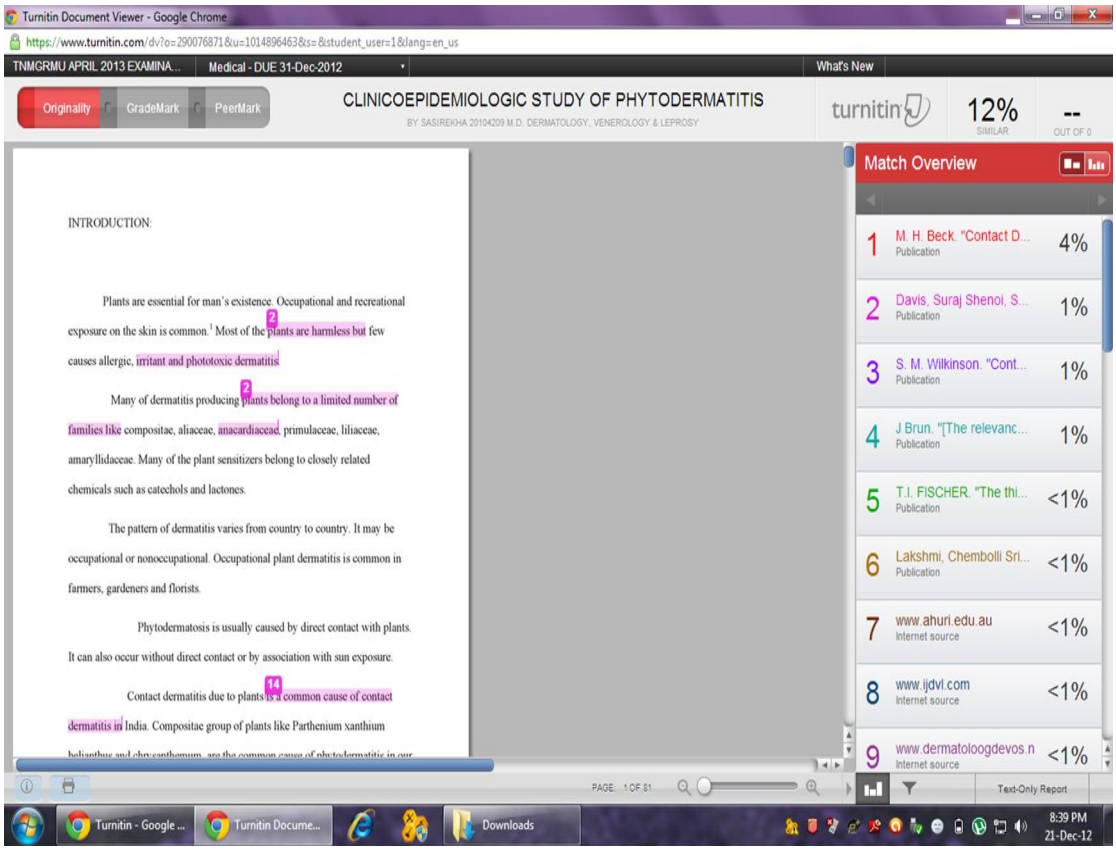
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INTRODUCTION:
Plants are essential for man's existence. Occupational and recreational exposure on the skin is common.[1] Most of the plants are harmless but few causes allergic, irritant and phototoxic dermatitis.
Many of dermatitis producing plants belong to a limited number of families like compositae, aliaceae, anacardiaceae, primulaceae, liliaceae, amaryllidaceae. Many of the plant sensitizers belong to closely related chemicals such as catechols and lactones.
The pattern of dermatitis varies from country to country. It may be occupational or nonoccupational. Occupational plant dermatitis is common in farmers, gardeners and florists.
Phytodermatosis is usually caused by direct contact with plants. It can also occur without direct contact or by association with sun exposure.
Contact dermatitis due to plants is a common cause of contact dermatitis in India. Compositae group of plants like Parthenium xanthium
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INTRODUCTION

Plants are essential for man's existence. Occupational and recreational exposure on the skin is common.[1] Most of the plants are harmless but few causes allergic, irritant and phototoxic dermatitis. Many of dermatitis producing plants belong to a limited number of families like Compositae, Aliaceae, Anacardiaceae, Primulaceae, Liliaceae, Amaryllidaceae. Many of the plant sensitizers belong to closely related chemicals such as catechols and lactones.

The pattern of dermatitis varies from country to country. It may be occupational or nonoccupational. Occupational plant dermatitis is common in farmers, gardeners and florists.

Contact dermatitis due to plants is a common cause of contact dermatitis in India. Compositae group of plants like Parthenium , Xanthium, Helianthus and Chrysanthemum are the common cause of phytodermatitis in our country. The Toxicodendron group (poison ivy, oak and sumac) produces both occupational and nonoccupational dermatitis. In Europe, Primula obconica is the principal cause of nonoccupational plant dermatitis.

Patch testing is a useful diagnostic tool in contact dermatitis. But patch testing is being carried out only in a few institutions in India, either with European series or indigenous allergens. However, the Indian Standard Series of 24 allergens approved by Contact and Occupational Dermatoses Forum of India (CODFI) has recently been made available.

REVIEW OF LITERATURE

CONTACT DERMATITIS

Contact dermatitis is an eczematous dermatitis caused by exposure to exogenous substances. It is classified as

- Irritant contact dermatitis
- Allergic contact dermatitis
- Photoallergic and phototoxic dermatitis
- Non eczematous dermatitis

IRRITANT CONTACT DERMATITIS:

Irritant contact dermatitis is a non immunological local inflammatory reaction which occurs as a result of a local toxic effect when the skin comes in contact with irritant chemicals. It is the commonest cause of contact dermatitis responsible for 80% of cases. Irritants produce a wide range of responses in skin. Irritant contact dermatitis may be [2]

- Acute irritant contact dermatitis
- Delayed acute Irritant Contact Dermatitis
- Irritant Reaction
- Chronic Irritant Contact Dermatitis

- Asteatotic Dermatitis
- Traumatic
- Acneiform
- Non erythematous irritation
- Non immune contact urticaria
- Subjective irritant responses

ALLERGIC CONTACT DERMATITIS:

Allergic contact dermatitis is a delayed-type hypersensitivity response to exogenous allergens which requires prior sensitization of the individual to that particular allergen.

PHOTOALLERGIC AND PHOTOTOXIC CONTACT DERMATITIS:[3]

Photoallergic reaction is a delayed hypersensitivity reaction in which photoallergic substances like sunscreens, fragrances, NSAIDS, quinolones, sulphonamides etc gets activated with UV radiation.

Phototoxic reactions are non allergic cutaneous reaction induced by various topical and systemic drugs.

NONECZEMATOUS DERMATITIS:

Contact dermatitis are usually eczematous but occasionally it may be noneczematous and can present as follows,

Contact urticaria

Erythema multiforme-like

Purpuric contact dermatitis

Lichenoid contact dermatitis

Lymphomatoid eruptions

Pigmented contact dermatitis

Leukoderma

Contact granulomatous

Onycholysis

HISTORY:

In 1906, von Pirquet coined the term ‘allergie’.[4]

Bloch and Steiner-Woerlich first proved allergic sensitization by using *Primula* extract on humans.

The term "phytophotodermatitis" was coined by Klaber in 1942.[5]

PATHOGENESIS OF ALLERGIC CONTACT DERMATITIS:

As classified by Coombs and Gel, allergic contact dermatitis is a type IV hypersensitivity reaction. Landsteiner and Chasel in 1950 firmly established allergic contact dermatitis as a form of cell mediated hypersensitivity. It is a T cell mediated cutaneous immune response to low molecular weight chemicals termed haptens.

The two main processes involved in the pathogenesis are

(A) Sensitisation

(B) Elicitation

A. SENSITISATION:

The main events are

(1) Binding of allergens to skin components:

Low molecular weight hapten chemicals penetrate the skin and associate with major histocompatibility complex (MHC) class II molecules present in epidermal dendritic cells and langerhans' cells. This occurs 6 hours after exposure to allergen.

(2) Recognition of 'complete' or conjugated antigen:

According to danger model, sensitization to MHC bound antigen requires co stimulatory factors like TNF alpha, GM-CSF and IL-1 which

are produced by keratinocytes.[6] Langerhans' cells travel through the afferent lymphatics to the paracortical areas of the regional lymph nodes, where apposition to T lymphocytes and antigen recognition occurs. Factors interfering with antigen presenting function of Langerhans cells include topical and systemic glucocorticoids, UV radiation and acquired immune deficiency syndrome.

(3) Proliferation and dissemination of sensitized T lymphocytes:

With recognition of antigen, cytokines like IL-1 and IL-2 are released and induce proliferation of antigen-specific cytotoxic CD8 and CD4 lymphocytes.[7] Dissemination of T cells occurs and they interact with Langerhans' cells and residual antigen in the skin. Cutaneous lymphocyte-associated antigen facilitates the entry of lymphocytes into skin. CLA positive lymphocytes express CCR 10, the receptor for CCL27 chemokine of basal keratinocytes causing localization to the site of sensitization.

B. ELICITATION:

The elicitation of allergic contact dermatitis requires close interactions between infiltrating T cells and antigen-presenting cells, which either reside in the skin or migrate from blood. If a sensitized individual is re-exposed to the specific allergen, reaction develops within 24 to 48 hours.

Sometimes a delayed elicitation response occurs following antigenic challenge in persons who are already sensitized. The reasons for delayed reaction time are low degrees of sensitivity, exposure to small amounts of allergen and delayed penetration of allergens.

PATHOGENESIS OF IRRITANT CONTACT DERMATITIS:

Penetration of the allergens through the permeability barrier cause mild damage to keratinocytes, and the release of mediators of inflammation with resultant T-cell activation. Once activation is initiated via epidermal cells, continuous T-cell activation independent of the exogenous antigen may be maintained. Tumor necrosis factor-α is a critical mediator, which, in addition to interleukins 6 and 1β upregulates expression of intercellular adhesion molecule-1 (ICAM-1). This is a predominant feature of irritant contact dermatitis.

FACTORS INFLUENCING CONTACT DERMATITIS:

INDIVIDUAL FACTORS:

(1) Constitution:

An individual's ability to get sensitized to allergens varies from person to person. The ability to quench free radicals, the level of antioxidant enzymes and the ability to form heat shock proteins may be genetically determined.

A TNF-α gene polymorphism has been demonstrated as a marker for susceptibility to irritant contact dermatitis.[8]

(2) Age:

Contact dermatitis is seen in individuals of all ages. Young children are prone for increased cutaneous penetration of allergens. Incidence of contact dermatitis is lower in individuals over 70 years of age. Young adults are more likely to have occupational or cosmetic allergies whereas elderly people are more liable to medicament.[9] Phytodermatitis commonly affects the age group ranging from 35 to 65 years.

(3) Sex:

Cell-mediated immune responses are stronger in women than men.[9] Many female-dominated occupations involve exposure to wet work. Female skin is more reactive in the premenstrual phase of the cycle.

(4) Race:

Racial differences are due to differences in exposure rather than predisposition.

(5) Medications:

Antihistamines and sodium cromoglycate appear to have little effect, whereas both prednisolone (dose >15 mg/day) and potent topical

steroids suppress allergic patch-test reactions.[10] Immunomodulators such as cyclosporine, azathioprine and UVB or PUVA therapy may also temporarily reduce contact allergic reactions .

(6) Atopy:

Atopic individuals have more easily irritated skins. Because of defective barrier, percutaneous absorption is increased in atopics.[11]

ENVIRONMENTAL FACTORS:

(1) Climate:

UVB exposure from the sun may depress the hypersensitivity and thereby reduce contact allergic reactions. Chapping of the skin during winter may act as precipitating factor for irritant contact dermatitis. Occlusion and increased sweating may predispose to contact dermatitis to shoes and clothes.

(2) Seasonal variation:

Allergenicity to *Primula obconica* varies with light and season. Compositae plants are destroyed by cold and frosty weather but regrow during the warmer spring and summer months.[12]

(3) Geographic variation:

Poison oak dermatitis is common in North America, while dermatitis due to *Primula obconica* is common in Europe. In India *Parthenium* is the commonest offending allergen and in Australia, wild Compositae plants are the main cause.

(4) Occupation:

Sensitisation to plant allergens is common among farmers, gardeners, florists, labourers, botanists.[12]

PHYTODERMATITIS

Phytodermatitis may be classified as –

I. Allergic sensitization
II. Irritant dermatitis
III. Phytophotodermatitis
IV. Pseudophytodermatitis

I. ALLERGIC SENSITIZATION

COMPOSITAE FAMILY

Asteraceae/Compositae (Daisy family) plants are common cause of phytodermatitis. The daisy family includes weeds, ornamental annuals, herbaceous perennials and vegetables.

More than 200 species have been reported to cause allergic contact dermatitis. *Parthenium hysterophorus, Chrysanthemum coronarium, Xanthium strumarium* and *Helianthus annus* are common sensitizers.

PARTHENIUM HYSTEROPHORUS

Parthenium hysterophorus belongs to the subfamily Asteraceae. It is a hybrid of *Parthenium confertum* and *Parthenium bipinnatifidum.* It is the most notorious compositae weed known to produce contact hypersensitivity. The other names are Congress grass, white top, carrot grass, wild fever few, ghajar ghas, bastard fever few. *Parthenium hysterophorus* was accidentally introduced in India in 1956 through imported food grains from USA.[12] The first case of Parthenium dermatitis was reported from Pune in 1968. Absence of natural restricting agents, high productivity level of the weed, efficient seed dispersal through wind and wide adaptability to varying soil and agro climatic conditions favors spread of the weed. Parthenium hysterophorus in India contains large amounts of sesquiterpene lactones.

Allergens:

Sesquiterpene lactones are the allergens in compositae plants. They include dehydrocostus lactone, alantolactone, costunolide and parthenolide. It is present in oleoresin fraction of leaf, stem, flower and pollen. Parthenium contains parthenin, hymenin, ambrosin and coronopilin.[12]

Clinical features:

Parthenium dermatitis primarily affects the exposed skin surface of adult male farmers and rural workers.

Parthenium dermatitis occurs predominantly in older males. It is rare in women and children. Male to female ratio is 5.5:1.26. This difference is because women have lesser exposure as compared to men but parthenium dermatitis without direct handling in areas of widespread growth of parthenium has been reported in housewives and indoor workers, suggesting that incidental exposure may sensitize a person to parthenium. Occurrence in teenagers and children is rare. Compositae mix sensitization occurs with higher frequency in atopic children than non-atopics.

In initial stages there is worsening of lesions during summer and monsoon with partial remission during winter but later the disease persists throughout the year with bouts of exacerbation.

Parthenium dermatitis primarily affects the hands, forearm, neck and face. Acute vesicular reaction can occur but the characteristically it is chronic and lichenified. In long standing cases air borne pattern changes to chronic reticuloid dermatitis.

Clinical patterns:

(1) Airborne contact dermatitis

(2) Chronic actinic dermatitis pattern

(3) Mixed pattern

(4) Exfoliative dermatitis

(5) Pseudophotodermatitis

(6) Atopic eczema like

(7) Hand eczema

(8) Localized dermatitis

(9) Photosensitive lichenoid dermatitis

(10) Prurigo nodularis like

(1) AIR BORNE CONTACT DERMATITIS:

Most of the airborne contact dermatitis starts from the eyelids, because the airborne allergens initially lodge over the skin folds and cause dermatitis. In the initial stages a seasonal variation is observed with flare up in summer and remission in winter. Later the dermatitis becomes persistent.[14]

Face, especially the eyelids, neck, ‘V’ area of the chest, and the elbow and knee flexures are commonly affected. It starts as an acute eczematous reaction. In sensitized individuals, the manifestations usually starts within 24 hours of exposure, but it may be delayed for up to 2-3 days or even longer in milder cases. In mild cases, dermatitis may manifest as only brief periods of erythema and itching, which subsides within a few hours or days. Moderate dermatitis is characterized by erythema, swelling, papules, or papulovesicles with itching and burning. Severe dermatitis may exhibit extensive vesiculation and exudation associated with edema. Repeated exposures over many years may result in widespread, extensive, and eventually chronic lichenified dermatitis that may persist throughout the year.

(2) CHRONIC ACTINIC DERMATITIS PATTERN:

It presents as lichenified papules, plaques or papulonodules over the exposed areas. Non-sun-exposed areas such as eyelids, retroauricular areas, under surface of chin, and depth of the skin folds are relatively spared. Patients with CAD pattern may develop exacerbation over flexures in the summers.

(3) MIXED PATTERN:

Here the features of both air borne contact dermatitis and chronic actinic dermatitis are present. It is considered as a transition phase from

classical ABCD to CAD pattern in natural history of parthenium dermatitis.[14]

(4) EXFOLIATIVE DERMATITIS:

It presents as diffuse involvement of the skin in the form of scaling, erythema, and induration.[15] There is often a past history of airborne contact dermatitis. Flexural lichenification may be seen.

(5) PSEUDOPHOTODERMATITIS:

In this pattern the exposed sites are involved. Here photoprotected sites like both eyelids, neck and retroauricular areas are also involved.

(6) ATOPIC ECZEMA LIKE:

This pattern mimics late onset atopic eczema with flexural accentuation of lesions.

(7) HAND ECZEMA:

Hand eczema is seen in gardeners after contact with the weed.

(8) LOCALIZED DERMATITIS:

In this pattern the lesions are confined to one or more localized areas.

(9) PHOTOSENSITIVE LICHENOID ERUPTION:

In this pattern the patients present with violaceous papules and plaques over the exposed areas.[16]

(10) PRURIGO NODULARIS-LIKE LESIONS:

It manifests as multiple hyperkeratotic papules and nodules over the extremities with characteristic histopathologic features similar to prurigo nodularis. There is usually a concurrent or a past history of active dermatitis.

The other rare clinical patterns reported are widespread dermatitis of non-airborne contact type, dermatitis of hands and feet, perianal dermatitis, vesicular hand eczema, seborrheic pattern [17] and dermatitis simulating lichen nitidus.[18]

CHRYSANTHEMUM CORONARIUM

Chrysanthemum, a genus of Compositae family of plants is grown as a decorative flowering plant throughout the world. Contact dermatitis to Chrysanthemum was first described by McCord et al in 1921.[19]

It is a common cause of occupational contact dermatitis among gardeners, florists, and horticulturists. Flowers, leaves and stem of the plant are sensitizers. Contact hypersensitivity to chrysanthemums can be associated with photosensitivity.

XANTHIUM STRUMARIUM

It is a composite weed which is a shrub growing to a height of 1.5 m. The common names are rough cocklebur, maruloomatum (Tamil), marulam athangi (Telugu), chotagokhru, kuthua (Hindi).[20] The origin of X. strumarium is North America. The flowering time in India is August – October. Xan1b, Xan vi a are the offending allergens.

Xanthium strumarium is also an important cause of plant dermatitis in India.[20] Airborne contact dermatitis due to Parthenium and Xanthium can coexist.[21] There have also been patients with allergy to Xanthium alone without concomitant allergy to Parthenium.[22]

HELIANTHUS ANNUS

It is commonly known as sunflower. Sesquiterpene lactones are present in capitate glandular hairs. The active chemical is 1-0-Methyl 4, 5 Dihydroniveusin.[23]

ALIACEAE

Alium cepa (Onion) and *Alium sativum* (Garlic) are common members of this family causing contact dermatitis. It commonly causes contact dermatitis in food handlers –housewives and chefs.

The allergens are present mostly in outer parts of bulbs. They have both irritant and allergenic properties. These compounds are derived from

various sulphur containing amino acids. Diallyl disulphide 5% is a suitable preparation for investigating garlic dermatitis.

The typical features of contact dermatitis to plants of the Aliaceae family are hyperkeratosis, scaling, fissuring and erythema on palmar aspects of tip of thumb, index and middle fingers. Other rare features are chelitis,[24] photoallergic contact dermatitis and systemic contact allergy.[25]

ANACARDIACEAE

Toxicodendron species, Anacardium, Mangifera, Semecarpus are members of this family. Derivatives of catechols mainly pentadecylcatechols, phenols, resorcinol and salicylic acid are the main allergens.

TOXICODENDRON PLANTS:

Poison ivy and poison oak are common causes of plant dermatitis in USA. Dermatitis is produced by exposure to bruised plant, characterized by streaky rash with erythema, papules, vesicles and bullae on the exposed sites. The bullae heal leaving pigmentation that persist for a long time which has a diagnostic value.[26]

ANACARDIUM OCCIDENTALE:

The common name is cashew nut. The oil from the shell of cashew nut contains a potential sensitizer which produces a reaction resembling poison ivy dermatitis.[27]

MANGIFERA INDICA:

Contact dermatitis is caused by oleoresin of mango tree sap or skin of the fruit. It causes extensive reaction on the fingers, back of forearm, and genitals among the pickers. Erythema, swelling and vesicles on the lips and vesicular rash on face can occur on eating.[28]

SEMICARPUS ANACARDIUM

The marking nut tree produces a black tarry oleoresin used to mark laundry. This blank ink is a potent sensitizer.[28]

PRIMULACEAE

Primula obconica is the leading cause of plant dermatitis in UK. The allergen is Primin. Its highest concentration is present in flowers' calyx. It produces mild transient erythematous patches and streaky vesicular plaques.[28]

ALSTROEMERIACEAE

Alstroemeriae (Peruvian lily) is commonly used in flower arrangements. The allergen is Tuliposide A. It produce finger tip dermatitis, chronic pulpitis amongst floriculturist and even depigmentation may occur.[28]

LILIACEAE

Tulipa and Hyacinthus are common offenders. Tulipaline A is the offending allergen. It is characterized by dry, fissured and hyperkeratotic lesions on fingertips. Face, cheeks and even genitalia can be affected.[28] Hyacinthus dermatitis is usually irritant in nature.

CACTACEAE

Most reactions are irritant but some are allergic. The sites of predilection are hands and finger webs. It is characterized by vesicles, papules, crusts and even ulceration.

AMARYLLIDACEAE

Narcissus and Galanthus are common offenders. Narcissus bulbs contain highly irritant needle shaped calcium oxalate crystals. Allergens are also produced but not yet isolated. It presents as papular rash and scaly erythema. Hand and forearm are commonly affected.[28]

II. IRRITANT DERMATITIS

Irritant contact dermatitis from plants is commonly divided into mechanical irritant contact dermatitis and chemical irritant contact dermatitis.

MECHANICAL IRRITANT CONTACT DERMATITIS:

Mechanical irritation may be produced by spines, thorns, specialized bristles and hairs.[29] Sharp trichromes of some cereals and grasses produce urticarial papules. Spicules of palms and cacti produce dermatitis. The prickly pear cactus has barbs (glochidia) which enter the skin producing a dermatitis resembling scabies.

CHEMICAL IRRITANT CONTACT DERMATITIS:

Chemical irritation may occur from contact with fluids or crystals in specialized hairs or other portions of the plant. Euphorbiaceae plants are common examples.[29] Buttercup (Ranunculus species) is an important irritant. Protoanemonin formed by breakdown of a glucoside in injured plant produce blisters. Sinigrin present in mustard and radish family is an irritant in presence of water. Bromelin in pineapple juice is an irritant.

III. PHYTOPHOTODERMATITIS

Photosensitization contact dermatitis due to plants is caused by photosensitizing compounds related to furocoumarin like xanthotoxin, bergapten, psoralen.[28] To initiate phytophotodermatitis, contact with a sensitizing furocoumarin and subsequent exposure to UV radiation (sunlight) is needed. Plants of Umbeliferae, Rutaceae and Moraceae family like angelica, celery, citron, figs, giant hogweed and lemon are common photo sensitizers.

5-methoxypsoralen present in the oil of Bergamot produces Berloque dermatitis. Phytophotodermatitis due to meadow grass is known as dermatitis bullosa striata pratensis.[30]

IV. PSEUDOPHYTODERMATITIS

Pseudophytodermatitis is an eruption that appears to be due to plants but in reality is produced by arthropods infesting the plants or by dyes and waxes applied to the skin of fruits and plant insecticides. The grain itch mite produce a generalized eruption of petichiae, wheals, vesicles and pustules.[31] Flour mite produces papular dermatitis. Cheese mite produces pruritic papular eruption. Azo dyes applied to skin of oranges and grapes cause dermatitis. Plant insecticides can cause dermatitis. Caterpillar hairs contain a toxin causing urticarial papular eruption.

INVESTIGATIONS

PATCH TEST

The patch test is at present the only practical test for demonstrating contact type of allergy. It is a specific proof of allergic contact dermatitis. The aim of patch test is to decide whether the test is positive or negative, whether it is an allergic reaction or as an irritant reaction and finally to quantitate the degree of sensitivity.

INDICATIONS:

(1) Eczematous disorders where contact allergy is suspected or is to be excluded

(2) Eczematous disorders failing to respond to treatment as expected

(3) Chronic hand and foot eczema

(4) Persistent or intermittent eczema of the face, eyelids, ears and perineum

(5) Varicose eczema

(6) To determine the actual allergens among many substances that is clinically suspected.

(7) As a predictive test to determine what materials the patient can safely tolerate.

When it is necessary to exclude the presence of contact dermatitis a negative patch test will help to support a clinical diagnosis other than allergic contact dermatitis.

CONTRAINDICATIONS:

(1) Acute dermatitis

(2) Immunosuppressive drugs such as systemic corticosteroids, cyclophosphamide, methotrexate etc, can suppress cell mediated hypersensitivity and cause false negativity. If the patient is on systemic corticosteroids upto a dose of 20mg prednisolone a day, the corticosteroid should be withdrawn completely on the day of application of the patch test till the reading is taken.[32]

(3) Application of topical steroids at the site of patch testing.[33]

(4) Pregnancy.

SELECTION OF THE PATCH TEST SITE:

Patch tests are generally done on the back, because

(1) It provides a large skin area for testing

(2) Pressure on the back during lying down helps a better contact of the antigen with the skin

(3) Least mobile area

(4) Less hairy

(5) Easy to do and read patch test

Other sites are: upper arm and thighs, less commonly flexural surface of the forearm and abdomen.

VEHICLE USE

Petrolatum is the best diluent, because it is more stable and nonallergenic.[34] Rarely allergy to petrolatum can occur.[35] Water and organic solvents slowly evaporate and alter the concentrations. When organic solvents are used a few minutes should be allowed for them to evaporate before they are applied to the skin which in turn prevents an irritant reaction. Ethyl Alcohol is the most commonly used organic solvent. Polypropylene syringes (without rubber plunges) are best for petrolatum. For liquids, glass dropper bottles are used.

ANTIGEN USED:

Defined SQLs mix is used for the screening of compositae allergy. It consists of a 0.1% mix of equimolar concentrations of 3 different SQLs (alantolactone, costunolide, and dehydrocostuslactone).[36] The acetone extract is preferred over aqueous extract.[37] Patch testing done with parts of fresh, frozen, and dried plants can produce false positive irritant reactions and even sensitization.

PATCH-TEST DOSE:

If petrolatum is used as the vehicle and disposable syringes are the containers, a length of 5 mm of test substance in vehicle will suffice. For a Finn chamber, 20 mg of allergen as a petrolatum dispersion has been shown to be the optimum dose.[38] 15 μL is the optimum dose if the vehicle is a liquid.

TYPES OF PATCH TEST:

1) The Standard Patch Test.

2) Open Patch Test

3) Delayed Occlusion Patch Test

4) Photopatch Test

5) Repeat Open Application Test(ROAT)

6) Usage test

7) Thin layer Rapid Use Epicutaneous test(TRUE)

1) The Standard Patch Test:

Here the antigen are applied on the skin of the patient and kept occluded for approximately 48 hours.

Patch test chambers:

Various patch test chambers recommended by International Contact Dermatitis Research group are:

a) Finn chamber

b) AL-test Unit

c) Duhring chamber

d) Van der bend square chamber

Finn chamber

Finn chamber was devised by Pirila (1975). The chambers are made up of stiff aluminium and have a diameter of 8mm and a depth of 0.5 mm.[39] A filter paper is required when testing with solutions.

AL-test unit

Fregert (1972) introduced the AL-test unit which was recommended by International Contact Dermatitis Research Group and

the North American Contact Dermatitis Group (1973). It consists of aluminium foil covered with polythene and 10mm central disc of filter paper adhered by heat and not by glue.[40]

Duhring chamber

Duhring chamber was designed by Frosch Kligman (1975) which is an enlarged aluminum unit measuring 18mm across and a capacity of 250 microlitres and six to eight can be fixed to the flexor surface of each forearm.[41]

Van der bend square chamber

Van der bend square chamber was first introduced by Malten and Nater et al (1976). A square application area makes it easier to differentiate between allergic test reaction and toxic reaction since the later corresponds exactly to the shape of chamber.

The adhesive tape is used not only to keep the tests in place, but also to provide some degree of occlusion with hydration of horny layer and better penetration. Some patients may be sensitive to colophony-based adhesive tape. An acrylic based or plastic based adhesive tape can alternatively be used in these patients.

An indigenous patch test method was described by Pasricha (1981). The unit consists of a 4cm square piece of adhesive plaster, at the

centre of which 4-8 layers of 2.5cm square piece of ordinary clean cotton gauze is stuck. One cm square piece of cotton or filter paper is placed in the centre of the gauze. Allergen is soaked into or placed on the central piece before placing the unit on the patient's skin.

The disadvantages of this chamber are :

1) The preparation is time consuming.
2) It occupies a large surface area, hence not ideal for testing more than 25 substances at one sitting.
3) Severe reactions may spread beyond the patch test site, because of lack of limiting device.

The antigen-impregnated-discs (AID) described by Pasricha (1981) can be used for patch testing by the patch test unit. An antigen-impregnated-disc consists of 1 cm square piece of Whatman-3 filter paper impregnated with a standard amount of the water soluble antigen. Antigen-containing-saucers (ACS) have the same principle as Finn chamber, but these already contain the antigen in the required amount.

Antigen-containing-saucers are made of an antigenically inert material and are 1cm in diameter and 0.5mm in depth and are filled with a standard amount of the antigen in ointment form.

These ready-made materials offer certain advantages. In the case of antigen impregnated discs :

1) The antigen discs are far more stable, being in the dried form.

2) There is no risk of increasing the concentration of the solutions by evaporation of the solvent.

3) There is no risk of contamination because each unit is an independent unit.

4) There is no need to measure the antigen solution for every test.

The antigen containing saucers also have the same advantages that there is no risk of contamination of the antigen because each antigen-containing- saucer is an independent unit and there is no need to measure the amount of the antigen ointment for each test.

An indigenous patch test unit resembling Finn chamber was described by Surinder Kaur and Sharma (1986). The unit was made from two items, adhesive tape and aluminum discs. The central discs of discarded aluminum vial-topes of uniform 7.0-7.5 mm size with smooth edges were placed on a piece of adhesive tape (12 x 5 cm) in two parallel rows of five each. The distance between the centers of adjacent discs was 2 cm on all sides.

For aqueous antigens, a wisp of cotton wool touched with the antigen was placed in the chamber with a forceps.

The advantages of this chambers over other indigenous units are ;

1) Having high value of ratio between volume / area; it gives a better response.

2) Need less time to prepare.

3) Tight apposition to the skin which is apparent from the indented ring on the surface when the unit is removed.

4) This can be washed and reused.

5) It occupies a small surface area, hence ideal for testing more than substances at one sitting.

6) Severe reactions may not spread beyond the patch test site, because of limiting device.

READING AND INTERPRETATION OF A PATCH TEST REACTION:

Patches are normally applied for 48 hours with readings taken 1 h after removal and again 48 h later.[42] The back is marked with indelible ink to identify the test sites. The patient should be instructed to avoid exercise, sweating and wetting the area.

Parameters to be observed:

- Erythema

- Infiltration (oedema),
- Fine structure-papule, vesicle, bulla, ulcer.
- Surface distribution of the reaction and area involved.

Recording is done according to International Contact Dermatitis Research Group. (From Wilkinson et al)

Grade	Structure	Interpretation
-		Negative
?+	Faint erythema only	Doubtful reaction
+	Palpable erythema, infiltration, possibly papules.	Weak positive reaction.
++	Erythema, infiltration, papules, vesicles	Strong positive reaction
+++	Intense erythema and infiltration and coalescing vesicles.	Extreme positive reaction
IR		Irritant reaction
NT		Not tested

Photopatch tests are graded similarly with a prefix ph.

DIFFERENCE BETWEEN IRRITANT AND ALLERGIC PATCH TEST REACTION

ALLERGIC REACTION	IRRITANT REACTION
Infiltration present .	Absent.
Homogenous reaction – papules, papulovesicles, coalescing vesicles.	Fine wrinkling, erythema and papules in follicular distribution, petichiae, pustules, bullae and necrosis.
Cover the test area homogenously.	Irregular, patchy, ring shaped or follicular in distribution.
Extrusion of the reaction beyond the tested area.	Limited to the tested area.

FALSE POSITIVE REACTIONS

1) Excess concentration of allergen
2) Substance tested is a primary irritant
3) Irritant vehicle
4) Impure substance
5) Uneven dispersion
6) Adhesive tape reaction
7) Current or recent dermatitis at the site
8) Angry back reaction[43]
9) Pressure effects
10) Artefact

FALSE NEGATIVE REACTIONS

1) Insufficient amount applied
2) Poor adhesion of patches
3) Inappropriate vehicles
4) Substance degraded
5) Reading performed too early
6) Pretreatment of site with topical steroids
7) Systemic immunosuppressant
8) UV irradiation of patch test site

COMPLICATIONS OF PATCH TEST :

1) Exacerbation of the pre-existing dermatitis
2) Generalized flare of dermatitis.
3) Spread of dermatitis from the patch test site
4) A focal flare-up of a previously patch tested site
5) Active sensitization
6) Hyper pigmentation or hypo pigmentation of the site

7) Pruritus

8) Folliculitis

9) Ulceration and scarring

10) Keloid formation

11) Systemic effects from absorption (e.g. Anaphylaxis)

MULTIPLE PATCH TEST REACTIONS

The causes of multiple patch test reactions are,

1) Non-specific hyperreactivity

2) Multiple primary hypersensitivities

3) Cross-reactions

1) Non specific hyper reactivity:

In active dermatitis, the uninvolved distant site has increased susceptibility to irritant reactions. Non-specific false-positive patch-test reactions may be induced by a strongly positive patch test reaction. These reactions are common with marginally irritant chemicals.

2) Multiple primary hypersensitivities:

Multiple primary hypersensitivities occur due to accumulation of several sensitivities in patients with dermatitis for a long time.

3) Cross-reactions:

It is a phenomenon where sensitization to one compound extends to one or more other compounds as a result of structural similarity. Cross reactivity can occur between the four compositae plants – Parthenium, Chrysanthemum, Helianthus and Xanthium.[44]

2) Open Patch Test:

Substances which are likely to produce irritant reactions under occlusion in the standard patch test, are required to be tested by the open patch test technique. In this test, the agent is painted on a small (1cm square) area of skin and the patient is instructed to avoid washing the area for at least 24 hours. The site is inspected 48 hours after the application and the reaction graded in the same way as for the standard patch test. To prevent the possibility of contamination of the antigen applied at one site with another antigen applied at an adjoining site, it is preferable to test only one agent at a time. In open patch the degree of sensitization is less because of the lesser penetration of allergens.

It is indicated:

1. If the testing substance is a potentially dermatitis inducing agent.
2. When testing a known irritant or sensitizer.
3. If organic solvents are tested.
4. If there is a risk of systemic effects from absorption

3) Delayed occlusion Patch Test:

Preparations which contain volatile substances, also can give rise to false positive reactions due to an irritant effect of the volatile components when occluded as during the standard patch test. For testing such agents, the substance should be applied on the skin and left exposed to air for ½ hour or so. During this period the volatile components will evaporate leaving behind the non volatile components. These should then be occluded as in standard patch test and the readings taken after 48 hours. This test therefore, is useful for testing the non-volatile components of the proprietary preparation.

4) Photopatch testing:

In patients with lesions in the exposed areas and who have worsening of lesions following sun exposure photo patch testing is done.[45] For this test, each substance is applied in duplicate patches. After 24 hours, one patch out of each pair is covered with a UV opaque material and the

other is irradiated with 5-10 J/sq.cm of UV-A. Psoralen plus UV-A fluorescent lamps are preferred. Readings are taken pre-irradiation, immediate post irradiation and 48 hours post irradiation.

The photopatch test is considered to be positive only if the test site exposed to the antigen and the light shows dermatitis, but there is no reaction at the unexposed patch test site, as also the skin area exposed to light only. Simply a more severe dermatitis at the photopatch site compared to the standard occluded patch test site is not sufficient to call it a positive photopatch test, because sunlight can non specifically aggravate almost any dermatitis reaction.

5) Repeated open application test (ROAT):

In this test, substances are applied twice daily for upto 4 weeks. The patient is advised to stop applying the test substance when a reaction is noticed. It is performed on the outer aspect of upper arm, antecubital fossa or scapular area of the back over an area of 5 cm^2.[46]

6) Usage test:

If the history suggests contact hypersensitivity but patch tests are negative, the patient is asked to use the preparation again. This test is useful in suspected cosmetic and clothing dermatitis.

7) Thin-layer-Rapid-Use-Epicutaneous test (TRUE Test):

It was devised by Fisher and Maibach (1985). It is a ready to apply test in which the test substances are incorporated in a flexible vehicle consisting of a thin layer of dried gel applied on a plastic, paper-plastic or aluminium foil backing.[47] This test produces an exact dosage, thin surface spread and high bioavailability of the allergens.

Other tests used:

Prick test

In this test the standard parthenium antigen or the plant material as such which is crushed and diluted with saline is used. Immediate reaction occurs at 15 mins and the late phase reaction occurs at 24-48 hours.[48]

RAST (Radio allergosorbent test)

This test can be done to detect parthenium specific antibodies but is less specific than prick testing.[48]

TREATMENT

Principles of treatment:

1) Avoidance of allergen
2) Treatment of dermatitis
3) Prevention of recurrences

1) AVOIDANCE OF ALLERGENS:

Avoidance of the causative plants is impractical in most set-ups; therefore prevention plays an important role in reducing the dermatitis. Working habits, hygienic measures and photoprotection are important measures in preventing allergic contact dermatitis.[49]

2) TREATMENT OF DERMATITIS:

Wet dressings with saline, potassium permanganate, aluminium acetate or silver nitrate are used for acute lesions .

Regular and liberal use of hydrating emollients and soap substitutes must be advised.

Corticosteroids are mainstay of treatment in parthenium induced dermatitis. Potent topical corticosteroids can be used for acute severe localised allergic contact dermatitis. In severe or widespread eruptions, systemic steroids may be used

Antibiotics are given for secondary infection.

Sedative antihistamines are given for pruritus.

Azathioprine is an effective steroid sparing agent. It is given in a dose of 100 mg daily with or without a 300 mg monthly bolus dose or as 300 mg bolus dose every week.[50]

Cyclosporine can be tried in recalcitrant cases.[51]

Methotrexate may be a useful alternative for patients with severe parthenium dermatitis.[52]

Oral hyposensitisation:

In oral hyposensitisation, the antigen is introduced into the body by a different route so that changes in the immune system occur. When the antigen is re-introduced through a normal route, the body does not develop clinical manifestations. It causes depletion of memory T-cells.[53]

Immunotherapy:

Immunotherapy with recombinant protein is administered in cases where patients are co sensitized with several unrelated pollen allergens. It has been reported useful in hay fever and allergic rhinitis and is under trial for use in ABCD. [36]

3) PREVENTION OF RECURRENCES:

Covering the exposed parts, removal of the allergen from the environment, or removal of the patient from the contaminated environment and desensitization methods can be tried to prevent recurrences. Drugs like azathioprine, methotrexate or cyclosporine can be used in maintenance doses.

AIMS OF THE STUDY

1. To find the common plant allergens causing phytodermatitis among patients attending our outpatient department.
2. To study the common age group affected by phytodermatitis.
3. To study the sex ratio among patients with phytodermatitis.
4. To find the occupational and non occupational causes of phytodermatitis.
5. To study the association of phytodermatitis and atopy.
6. To study the association between the duration of occupational exposure required for clinical manifestation.

MATERIALS AND METHODS

STUDY

Prospective observational study

SAMPLE

100 cases of phytodermatitis who attended dermatology out patient department, Govt. General Hospital, Chennai from Oct 2010 – Sept 2012 who were patch test positive were included in the study. The study was approved by the Institutional ethical committee. A written consent form was signed by all the patients.

INCLUSION CRITERIA :

1. Clinically suspected patch test positive patients with phytodermatitis.
2. Patients who are able to understand the value of patch test, ready to give consent and can come for regular follow up are included in the study.
3. Patient with active dermatitis will be first treated and then subjected to patch testing to avoid false positive reaction.

EXCLUSION CRITERIA :

1. Patients who are immunocompromised due to disease or drugs.

2. Patients with contact dermatitis and who patch tested negative or had doubtful reactions for the suspected plant antigens.

METHOD

- A detailed history of the patients included in the study including age, sex, chief complaints, type of occupation and duration of exposure to suspected plant were noted.
- History of seasonal exacerbation of lesions was noted.
- History of atopy in self or family members, photosensitivity, drug intake, and past history of similar illness were noted.
- Morphology of lesions, site of involvement and clinical pattern were noted.
- Based on the type and nature of exposure to a particular antigen, the patients were patch tested with the appropriate antigens.

PROCEDURE

Patch test allergens which are to be used are approved by Contact and Occupational Dermatoses Forum of India(CODFI).

Antigens for the following plants obtained from creative drug industries, Mumbai were used

- Parthenium 15%
- Chrysanthemum 15%

- Helianthus annus 15%
- Xanthium 15%
- Garlic 100%

INSTRUCTIONS GIVEN TO THE PATIENTS

Patients were given information about allergic contact dermatitis, the aim of the test, the application and reading time and the reactions expected to develop.

The following instructions were given:

1. Patch test to be left in place for two days.
2. To avoid bathing, exercise or any other activity causing excessive sweating.
3. To avoid friction or scratching the test site.
4. To avoid tight underclothes.
5. To avoid exposure of the test site to sunlight/ UV light.
6. To report immediately if there is severe itching or irritation.
7. To come for patch test reading after 48 and 96 hours.

Patch testing was done as follows:

1. The protective foil of the Finn chamber was removed and the patch test unit was placed on the table with the Finn

chambers facing up.

2. The allergens were stored in refrigerator at 4-8 degree Celsius . 5mm length of the allergen from the syringe was put in the centre of the aluminium chamber.

3. The upper back of the patient was gently cleaned with sterile gauze before antigen application.

4. Allergens were applied on the right side of upper back and the control was applied on the left side parallel to the allergens on the right side.

5. Patches were removed after 2 days.

6. Readings were taken 1 hour after removal.

7. A second reading was taken on day 4.

The readings were interpreted according to the International Contact Dermatitis Research Group (ICDRG) guidelines.

OBSERVATIONS AND RESULTS

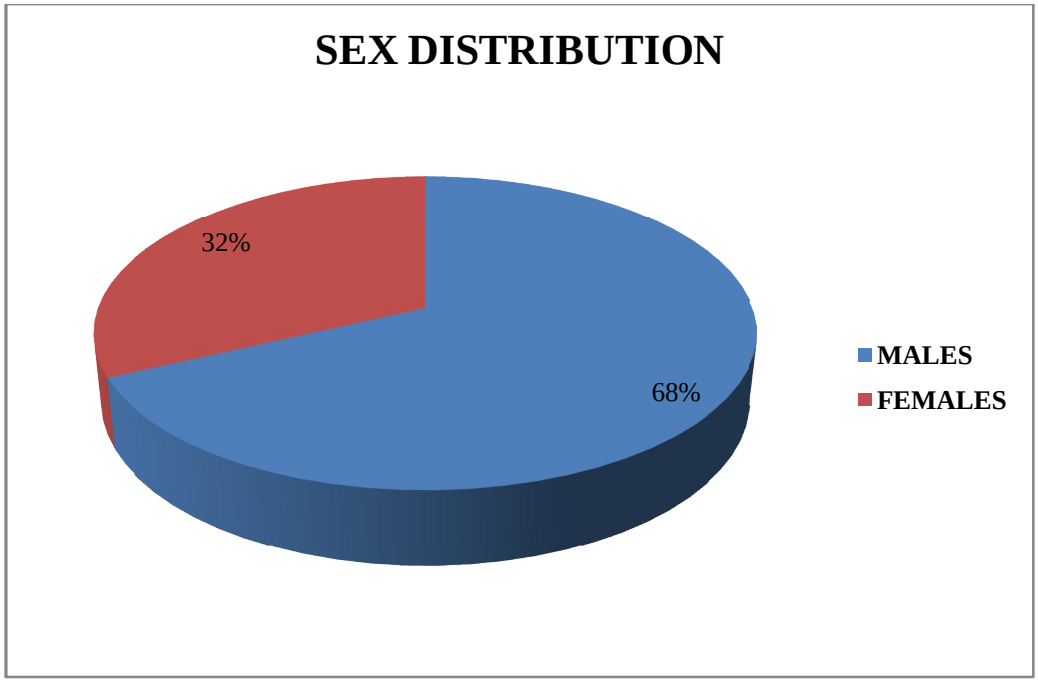
I. SEX DISTRIBUTION

TABLE 1

Distribution according to sex

Sex	Number of cases (N=100)	Percentage
Males	68	68
Females	32	32

Out of 100 patients analysed 68% were males and 32% were females. The male to female ratio is 2.1:1



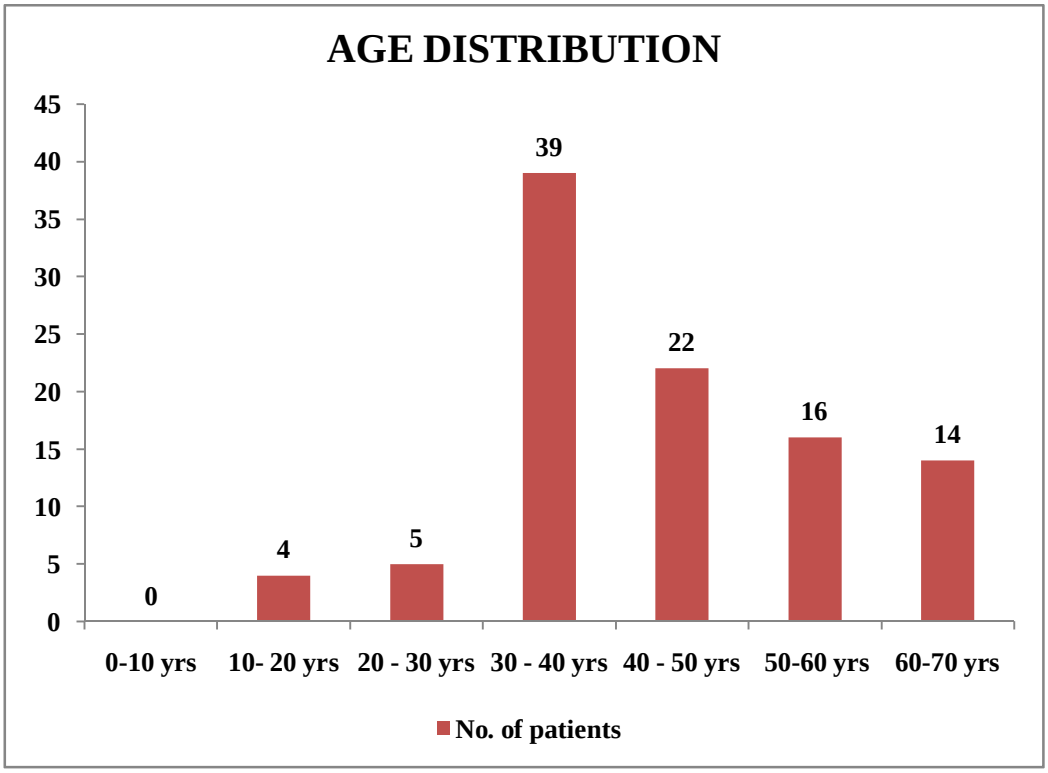
II. AGE DISTRIBUTION

TABLE 2

Distribution according to age

AGE	TOTAL	%
0-10 yrs	0	0
10-20 yrs	4	4
20-30 yrs	5	5
30-40 yrs	39	39
40-50 yrs	22	22
50-60 yrs	16	16
60-70 yrs	14	14

Patients in the age group 30 – 40 years were commonly affected. The mean age was 43.3 years.



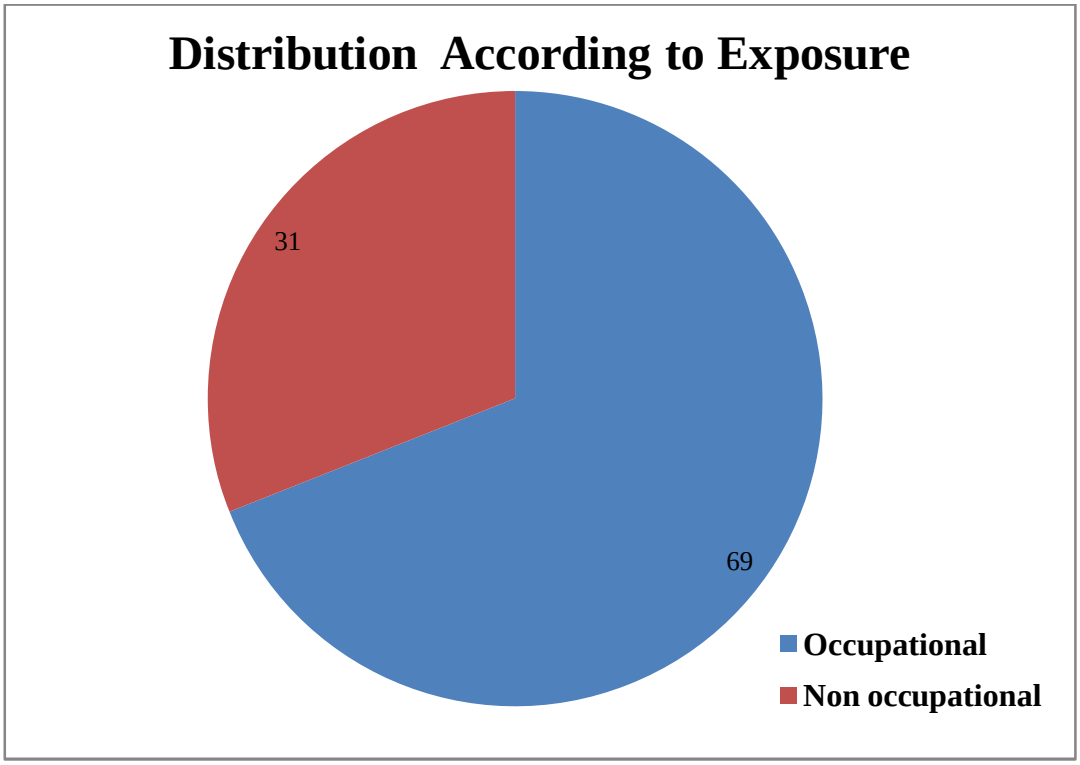
III. DISTRIBUTION ACCORDING TO EXPOSURE

TABLE 3

Distribution according to exposure

Type of exposure	No. of cases n = 100
Occupational	69
Non occupational	31

In this study occupational exposure was common (69%)



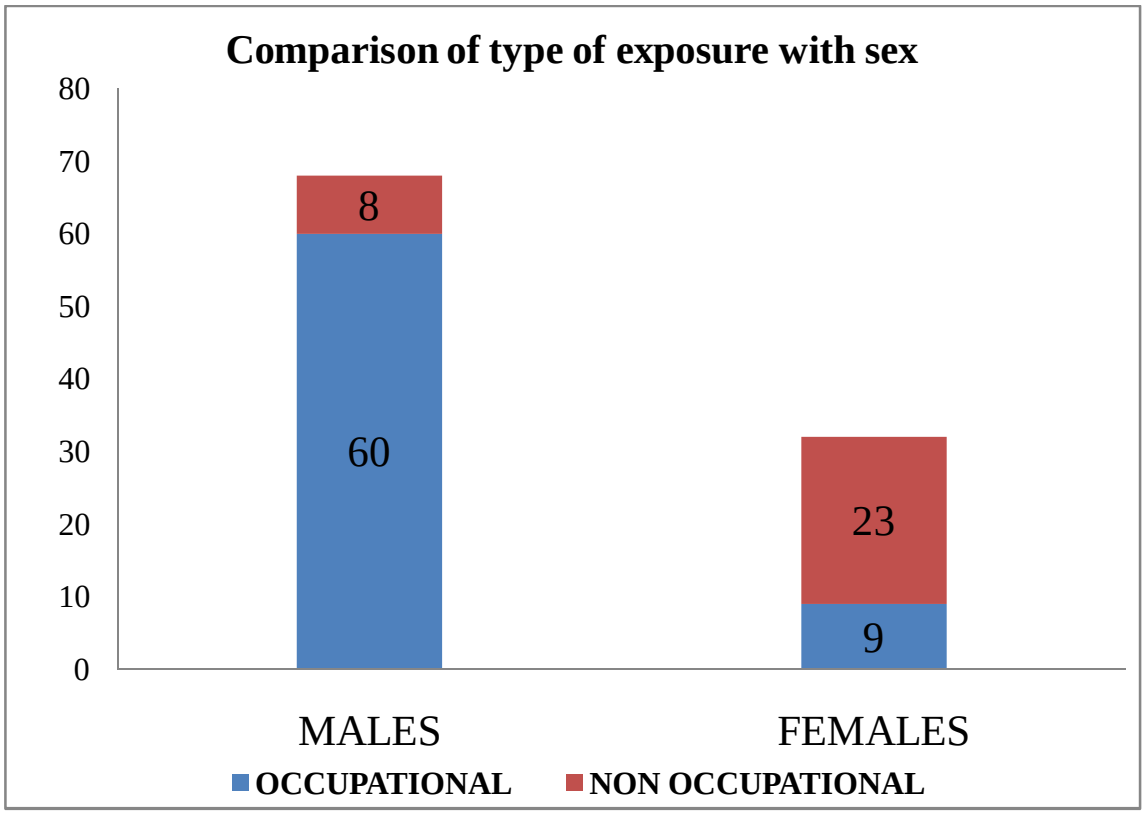
IV. COMPARISON OF TYPE OF EXPOSURE WITH SEX

TABLE 4

Comparison of type of exposure with sex

	Males	Females	Total
Occupational	60	9	69
Non occupational	8	23	31

Occupational type of exposure is common in males (88.23%) whereas in case of females non occupational exposure is common (71.87%). P value is less than 0.0001 which is statistically significant.



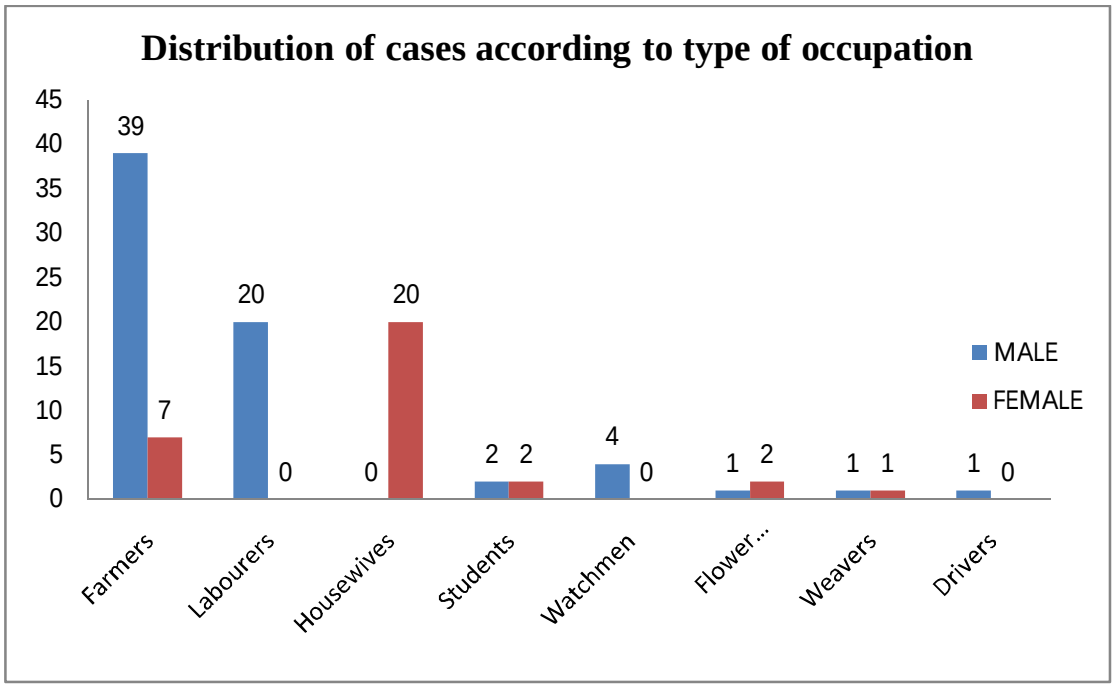
V. DISTRIBUTION ACCORDING TO OCCUPATION

TABLE 5

Distribution according to occupation

Occupation	Males	Females	Total	Percentage
Farmers	39	7	46	46
Labourers	20	-	20	20
Flower vendors	1	2	3	3
Household workers	-	20	20	20
Students	2	2	4	4
Watchmen	4	-	4	4
Weavers	1	1	2	2
Drivers	1	-	1	1

In this study most of them are farmers (46%) followed by labourers (20%), housewives (20%), students (4%), watchmen (4%), flower vendors (3%), weavers (2%) and drivers (1%).



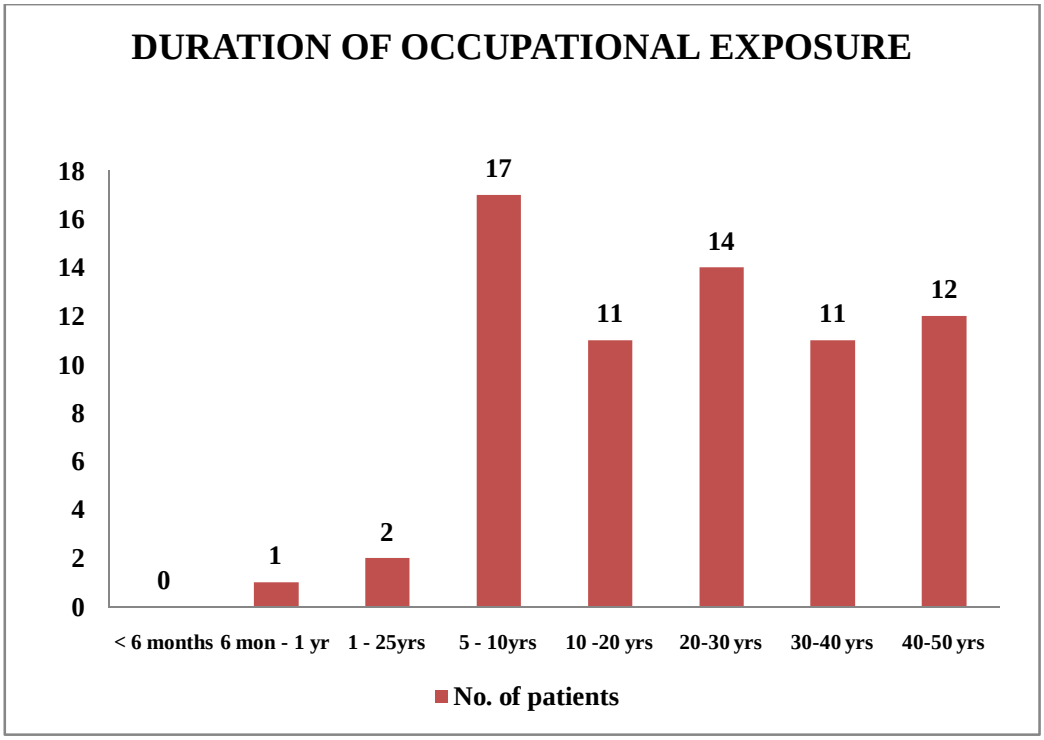
VI. DURATION OF OCCUPATIONAL EXPOSURE

TABLE 6

Distribution of cases according to duration of occupational exposure

Duration of occupational exposure	Number of cases	Percentage
< 6 months	0	0
6 mon – 1 yr	1	1
1 – 5 yrs	2	2
5 – 10 yrs	18	18
10 – 20 yrs	11	11
20- 30 yrs	14	14
30 - 40	11	11
40- 50 yrs	12	12

The duration of occupational exposure ranges from 6 months to 50 years and is commonly more than 5 years.



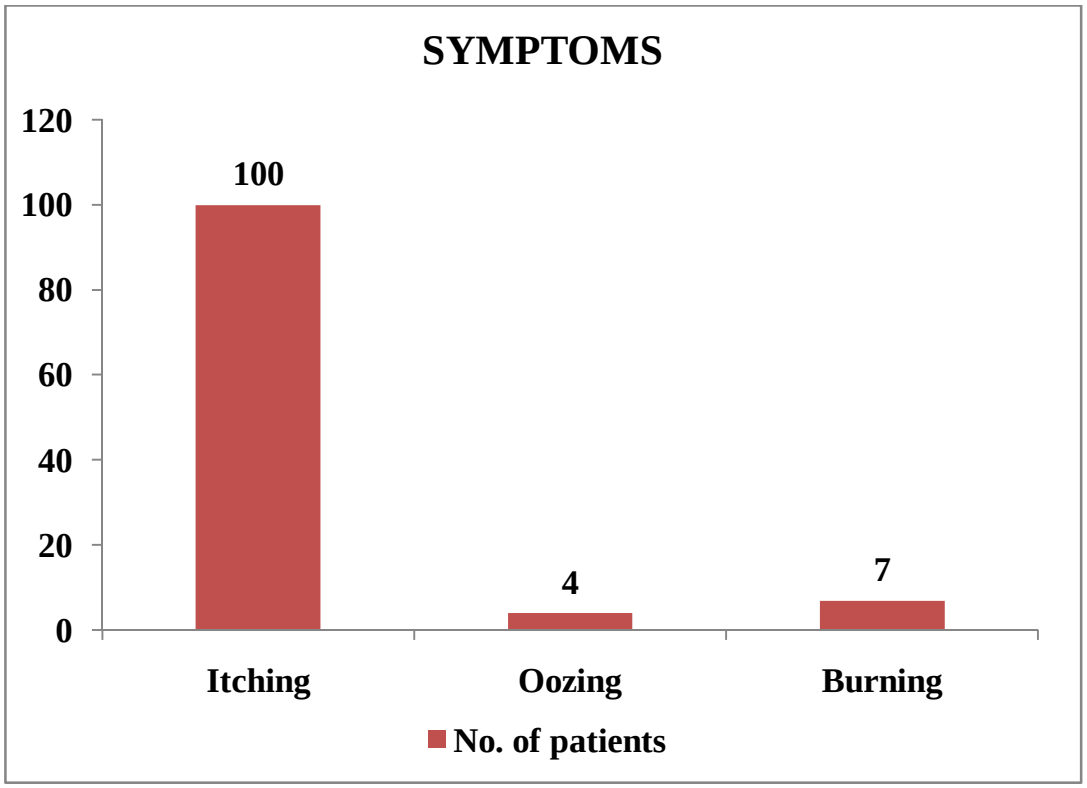
VII . SYMPTOMS

TABLE 7

Distribution of cases according to symptoms

SYMPTOMS	Number of patients	Percentage
Itching	100	100
Oozing	4	4
Burning sensation	7	7

In this study all the patients (100%) complained of itching, 4% had oozing and 7% complained of burning sensation.



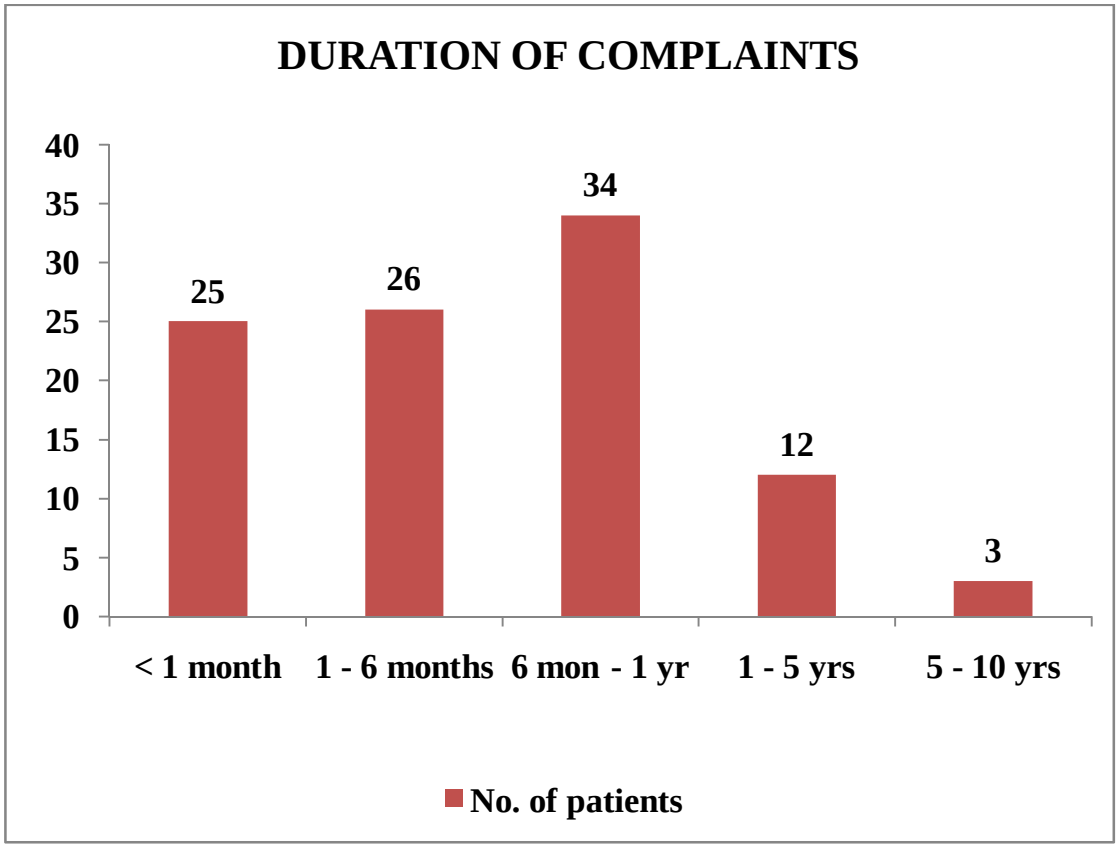
VIII. DURATION OF COMPLAINTS

TABLE 8

Distribution of cases according to duration of complaints

Duration of complaints	Number of cases	Percentage
< 1 month	25	25
1 – 6 months	26	26
6 mon – 1 yr	34	34
1 – 5 yrs	12	12
5 – 10 yrs	3	3

In this study 25% had symptoms for less than a month, 26% had symptoms for 1-6 months and 34% for 6 months to 1 yr.



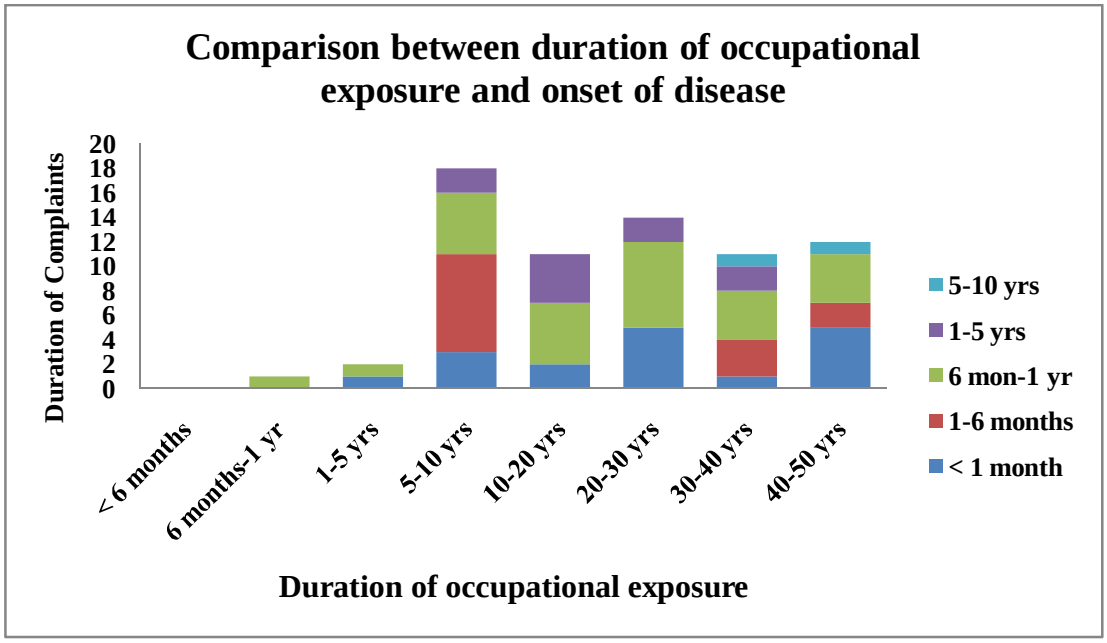
X. COMPARISON BETWEEN DURATION OF OCCUPATIONAL EXPOSURE AND ONSET OF DISEASE

TABLE 8

Comparison between duration of occupational exposure and onset of disease.

Duration of Occupational exposure	Duration of complaints at the time of presentation				
	< 1 month	1-6 months	6 mon-1 yr	1-5 yrs	5-10 yrs
< 6 months	0	0	0	0	0
6 mon-1 yr	0	0	1	0	0
1-5 yrs	1	0	1	0	0
5-10 yrs	3	8	5	2	0
10-20 yrs	2	0	5	4	0
20-30 yrs	5	0	7	2	0
30-40 yrs	1	3	4	2	1
40-50 yrs	5	2	4	0	1

This shows that there is no correlation between duration of occupational exposure and onset of disease.



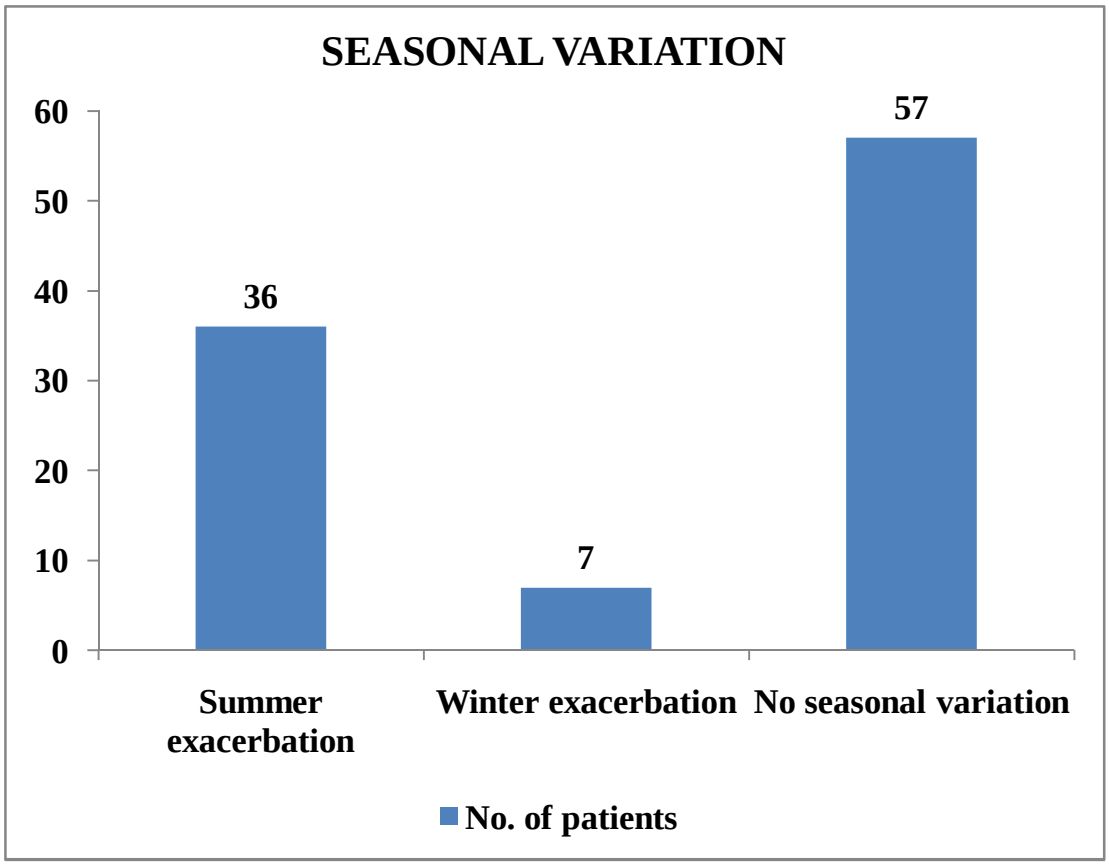
X. SEASONAL VARIATION

TABLE10

Percentage of cases showing seasonal variation

Season	Number of patients showing exacerbation of lesions	Percentage
Summer exacerbation	36	36
Winter exacerbation	7	7
No seasonal variation	57	57

In this study 57% had no seasonal variation and 36% had summer exacerbation.



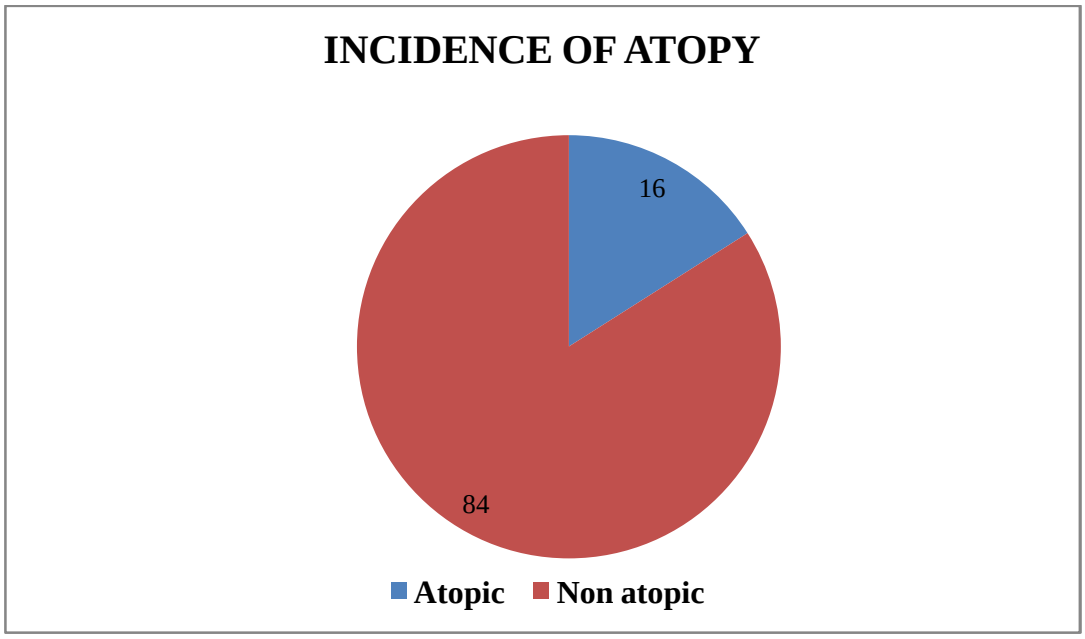
XI. ATOPY

Table 11

Incidence of Atopy

	No. of patients	Percentage
Atopic	16	16%
Non atopic	84	84%

In this study 16% were atopics.



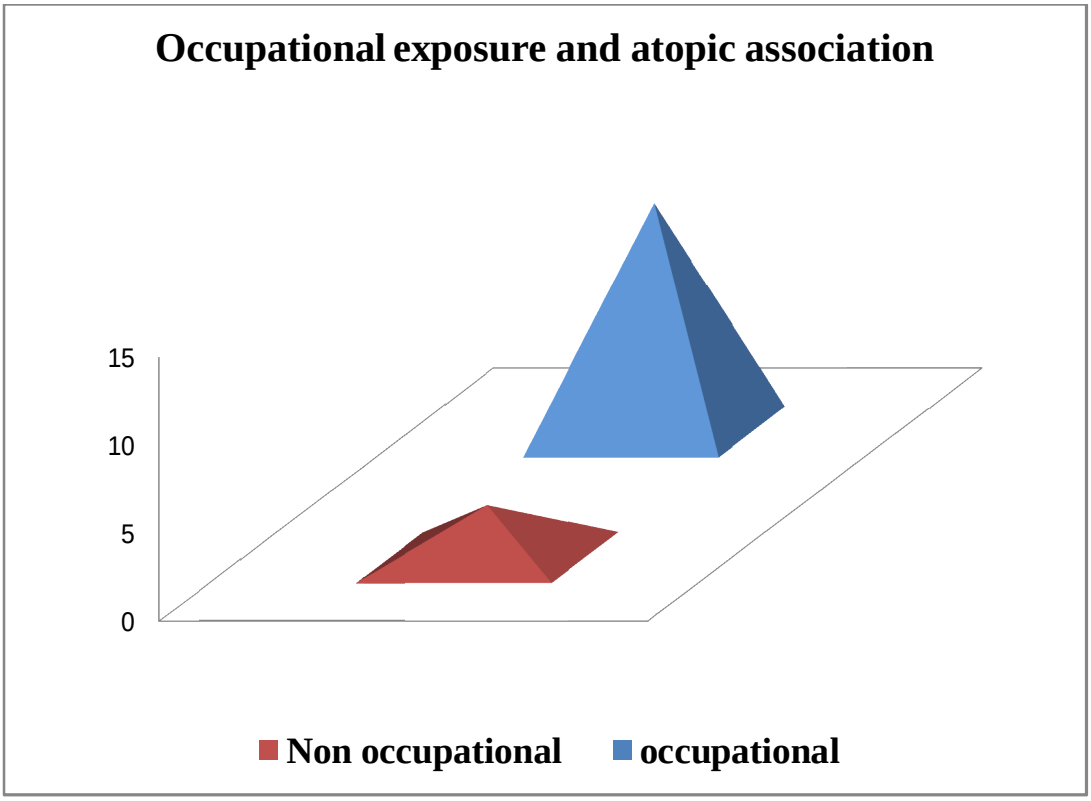
XII. OCCUPATIONAL EXPOSURE AND ATOPIC ASSOCIATION

TABLE 12

Occupational exposure and atopic association

Exposure	Atopy
Occupational	13
Non occupational	3

Among the 16 atopics, occupational exposure is common (81.25%).



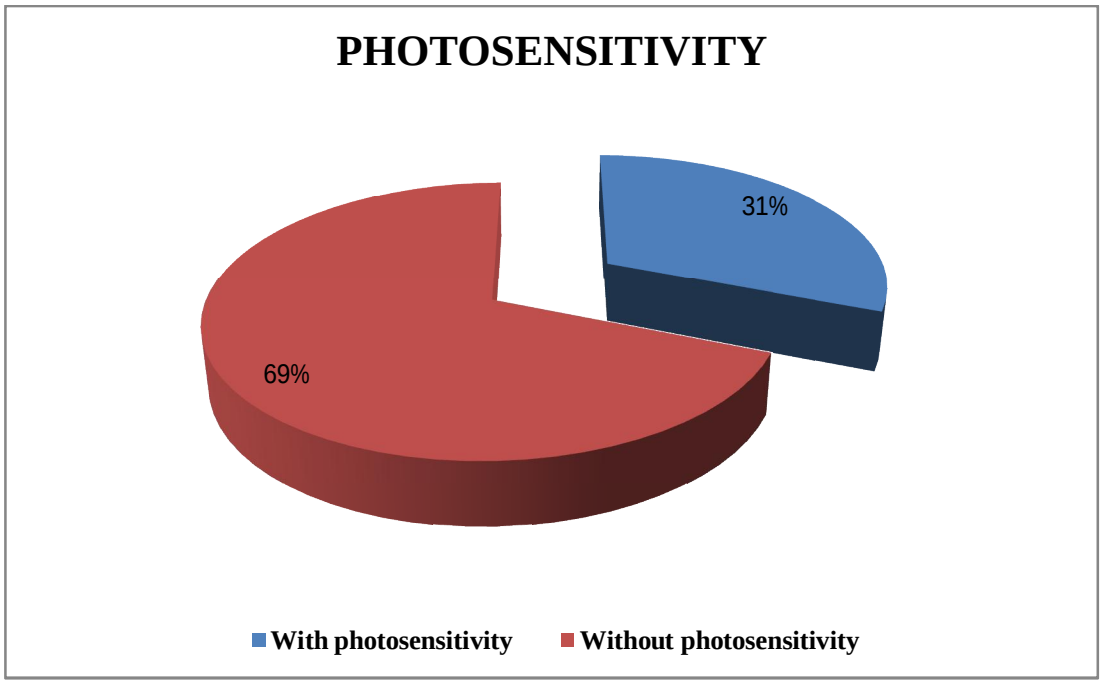
XIII. PHOTOSENSITIVITY

TABLE 13

Percentage of cases with photosensitivity

	No. of cases n = 100	Percentage
With photosensitivity	31	31%
Without photosensitivity	69	69%

In this study about 31% of patients gave history of photosensitivity.



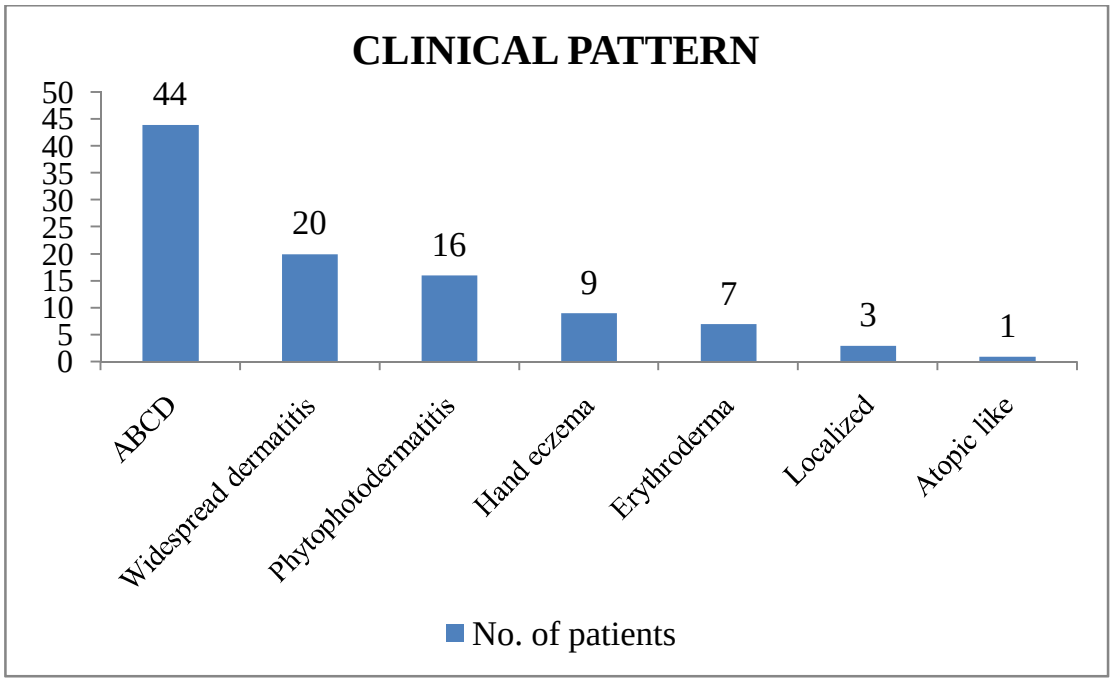
XIV. CLINICAL PATTERN

Table 14

Clinical pattern of plant dermatitis

Clinical Pattern	Number of patients	Percentage
ABCD	44	44
Widespread dermatitis	20	20
Phytophotodermatitis	16	16
Hand eczema	9	9
Erythroderma	7	7
Localized	3	3
Atopic like	1	1

In this study Air borne contact dermatitis was the most common pattern observed (44%), followed by widespread dermatitis (20%) and phytophotodermatitis(16%).



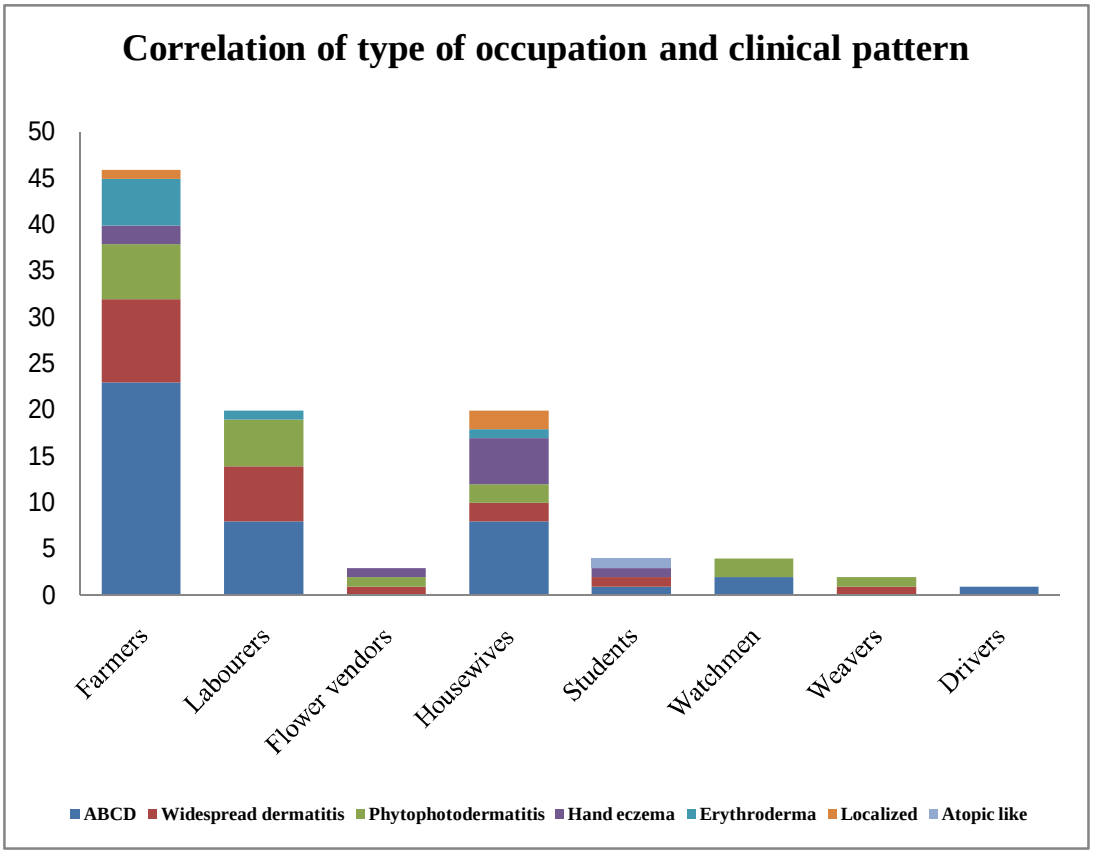
XV. CORRELATION OF TYPE OF OCCUPATION AND CLINICAL PATTERN

TABLE 15

Correlation of type of occupation and clinical pattern

Occupation	Clinical pattern						
	ABCD	**Widespread dermatitis**	**Phytophoto dermatitis**	**Hand eczema**	**Erythro derma**	**Loca lized**	**Atopic like**
Farmers	23	9	6	2	5	1	0
Labourers	8	6	5	0	1	0	0
Flower vendors	0	1	1	1	0	0	0
Housewives	8	2	2	5	1	2	0
Students	1	1	0	1	0	0	1
Watchmen	2	0	2	0	0	0	0
Weavers	0	1	1	0	0	0	0
Drivers	1	0	0	0	0	0	0

Among the farmers, ABCD is the commonest pattern followed by widespread dermatitis, phytophotodermatitis and erythroderma. Hand eczema is common among housewives.


Correlation of type of occupation and clinical pattern
50
45
40
35
30
25
20
15
10
5
0
Farmers
Labourers
Flower vendors
Housewives
Students
Watchmen
Weavers
Drivers
ABCD
Widespread dermatitis
Phytophotodermatitis
Hand eczema
Erythroderma
Localized
Atopic like

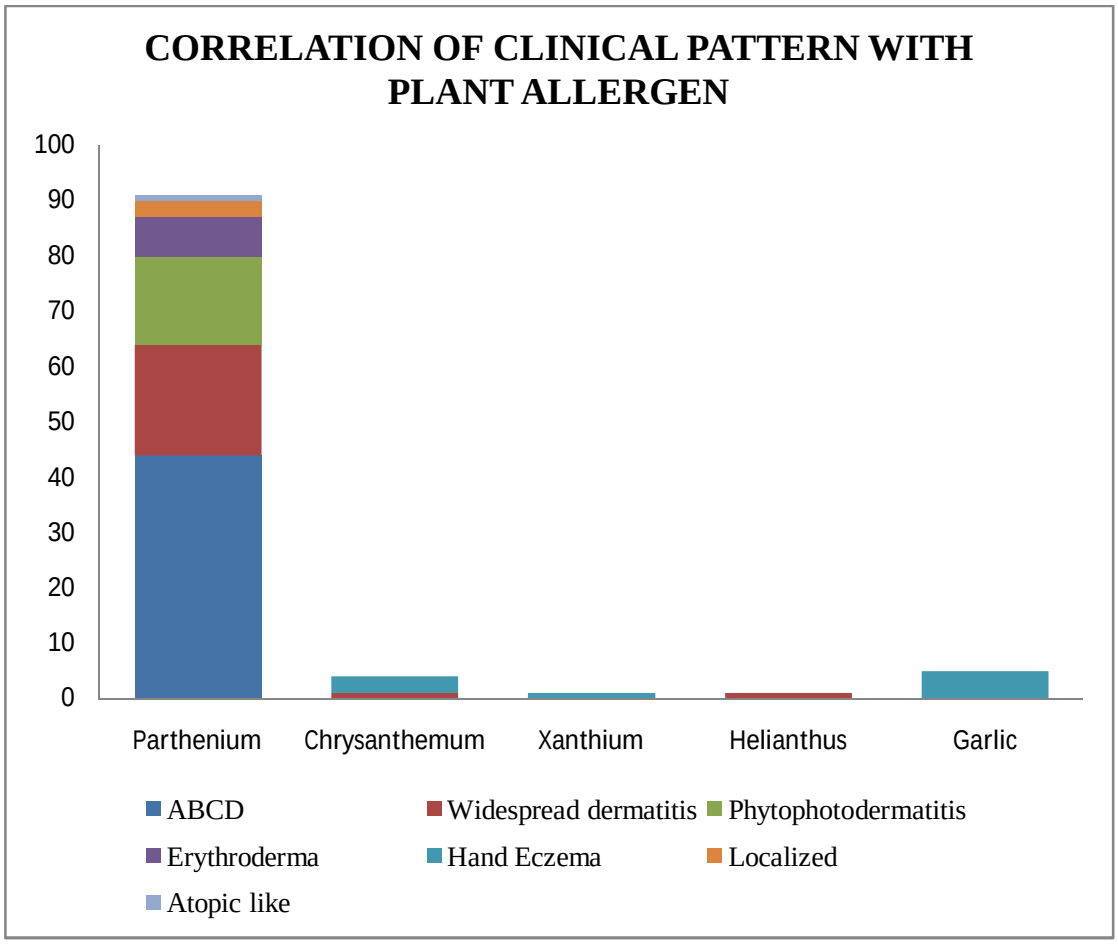
XVI. CORRELATION OF CLINICAL PATTERN WITH THE PLANT ALLERGEN

TABLE 16

Correlation of clinical pattern with the plant allergen

	Parthenium	Chrysanthemum	Xanthium	Helianthus	Garlic
ABCD	44	-	-	-	-
Widespread dermatitis	20	1	-	-	-
Phytophoto dermatitis	16	-	-	1	-
Erythroderma	7	-	-	-	-
Hand Eczema	-	3	1	-	5
Localised	3	-	-	-	-
Atopic like	1	-	-	-	-

In this study Air borne contact dermatitis was the commonest pattern in patients with Parthenium dermatitis (48.35%). Chrysanthemum dermatitis commonly presented as hand eczema(75%). All patients with contact dermatitis due to garlic presented as hand eczema.


CORRELATION OF CLINICAL PATTERN WITH PLANT ALLERGEN
100
90
80
70
60
50
40
30
20
10
0
Parthenium
Chrysanthemum
Xanthium
Helianthus
Garlic
ABCD
Widespread dermatitis
Phytophotodermatitis
Erythroderma
Hand Eczema
Localized
Atopic like

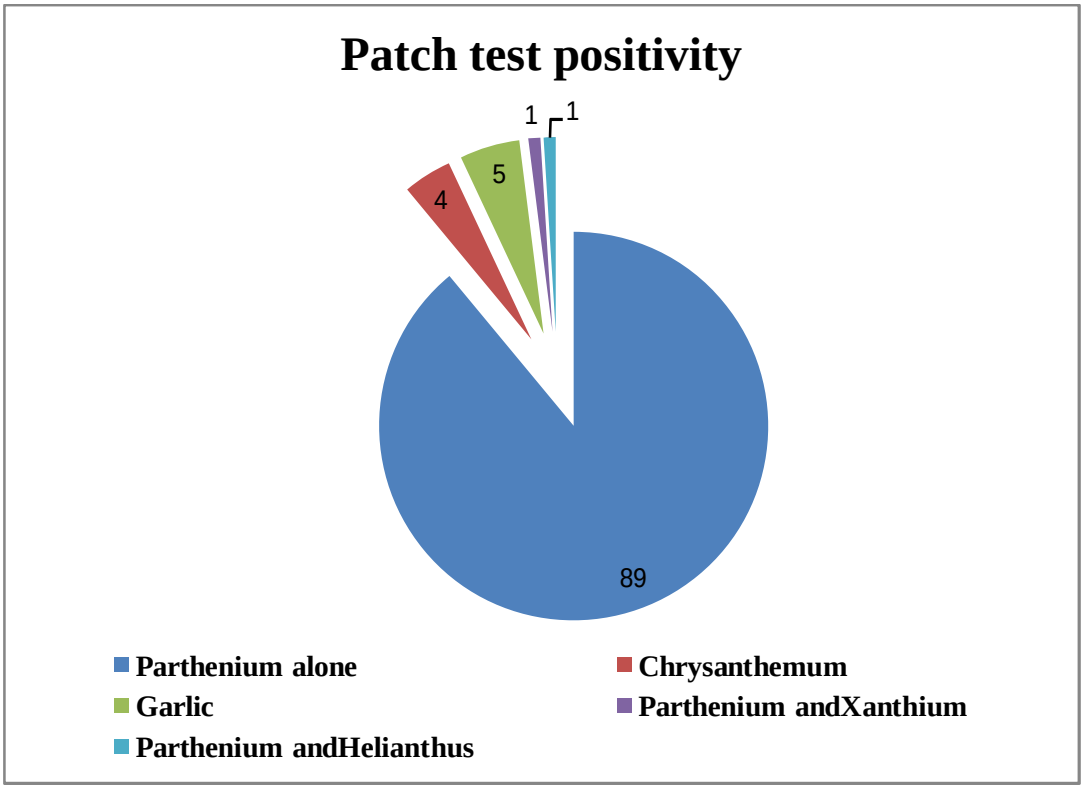
XVII . PATCH TEST POSITIVITY

TABLE 17

Percentage of cases showing patch test positivity to plant antigens

ALLERGEN	Number	%
Parthenium alone	89	89
Parthenium and Xanthium	1	1
Parthenium and Helianthus	1	1
Chrysanthemum	4	4
Garlic	5	5

In this study Parthenium was found to be the most common allergen (91%).



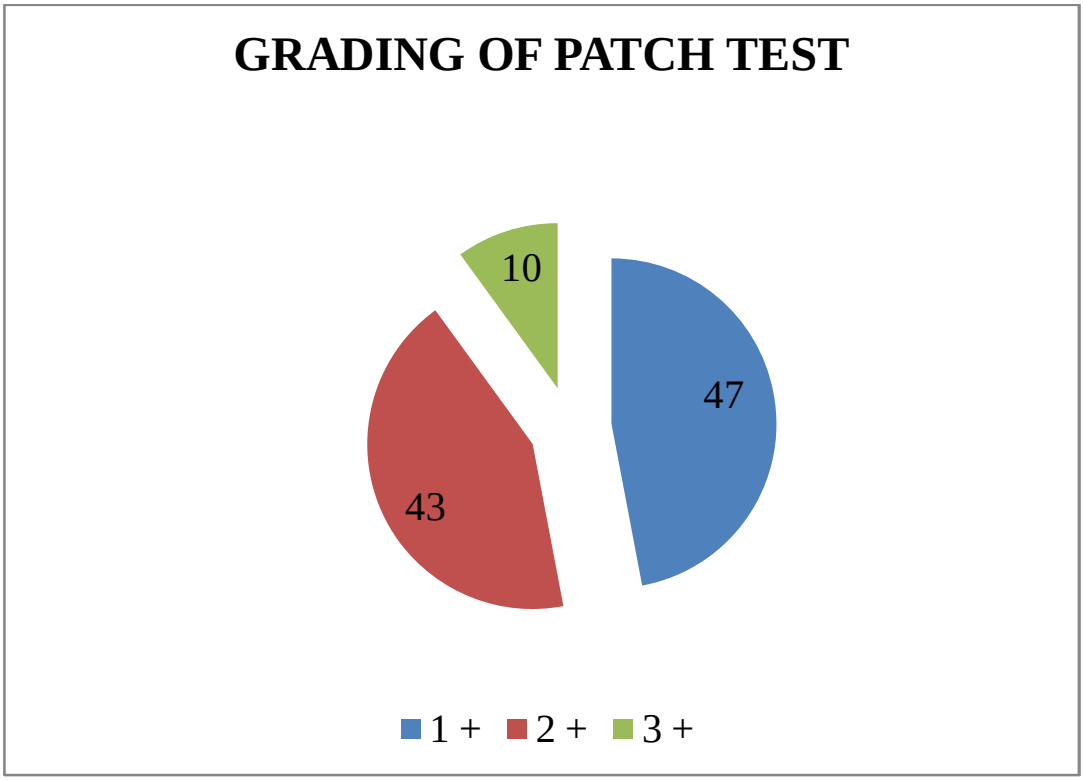
XVIII. GRADING OF PATCH TEST

TABLE 18

Distribution of cases according to grading of patch test

Degree of positivity	Number of patients (n=100)	Percentage
1 +	47	47
2 +	43	43
3 +	10	10
IR	0	0

In this study 1+ reaction was common (47%), 43% showed 2+ reaction and 3+ reaction was found in 10%.



ABCD DUE TO PARTHENIUM – INVOLVEMENT OF EYELIDS

ABCD DUE TO PARTHENIUM – INVOLVEMENT OF RETROAURICULAR AREA

ABCD DUE TO PARTHENIUM – INVOLVEMENT OF NECK

PHYTOPHOTODERMATITIS DUE TO PARTHENIUM – INVOLVEMENT OF EXPOSED AREAS

ERYTHRODERMA DUE TO PARTHENIUM

ATOPIC LIKE PATTERN DUE TO PARTHENIUM – FLEXURAL INVOLVEMENT

HAND ECZEMA DUE TO CHRYSANTHEMUM

PATCH TEST GRADING : +

PATCH TEST GRADING : ++

PATCH TEST GRADING : +++

PLANT ANTIGENS

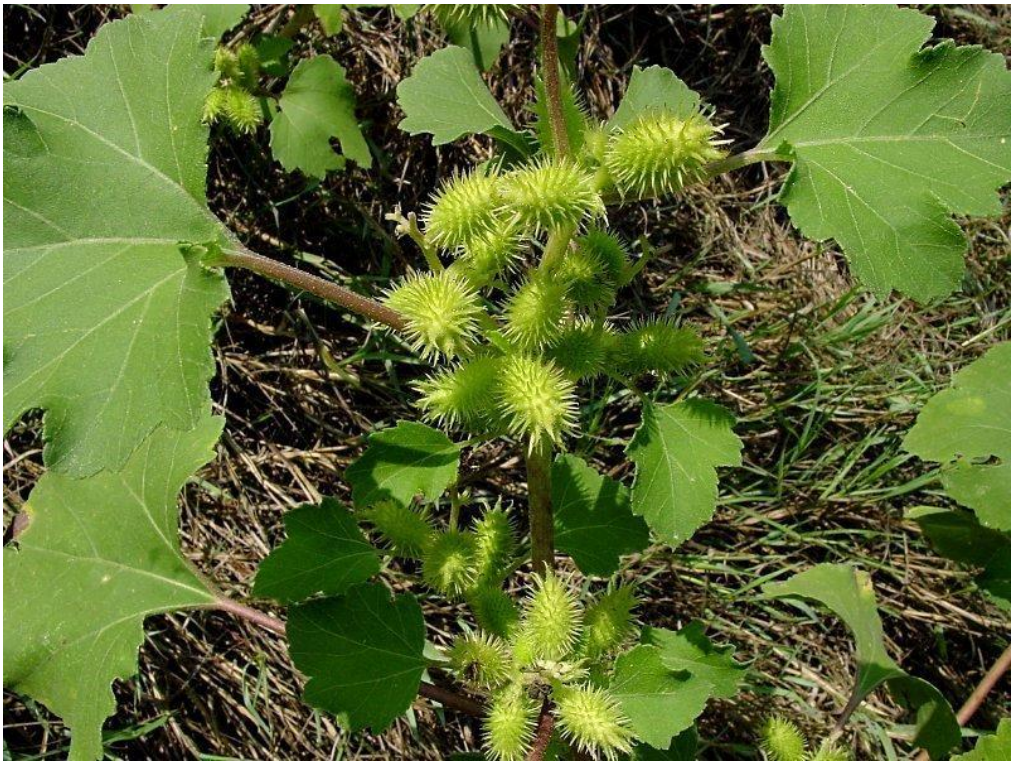
FINN CHAMBER

PARTHENIUM

CHRYSANTHEMUM

HELIANTHUS

XANTHIUM



SUMMARY

- In this study, males were commonly affected than females with a ratio of 2.1:1.
- Patients in the age group 30 – 40 years were commonly affected.
- Most of the patients were exposed occupationally
- Farmers predominated the occupational group and housewives predominated the non occupational group.
- Among the males, occupational exposure was common but in females non occupational exposure was common.
- The duration of occupational exposure ranged from 6 months to 50 years.
- The duration of complaints ranged from less than a month to 10 years.
- There was no correlation between duration of occupational exposure and onset of disease.
- About 16% of the patients had atopy.
- 31% of the patients gave a history of photosensitivity.
- Airborne contact dermatitis was the most common pattern observed.
- Parthenium was the most common allergen.

DISCUSSION

Contact dermatitis to plants accounts for 3% of patients among contact dermatitis. This condition presents with varied clinical features and the diagnosis and nature of causative agents is usually established only after an elaborate history, clinical examination of the patient and patch testing.

1. SEX:

The males outnumbered females in this study with a ratio of 2.1:1.

This may be because males take up more field work than females, where they come in contact with various plants.

In a study by VK Sharma et al the male:female ratio was 2:1.[54]

K Vinod Sharma et al conducted a study on contact dermatitis due to plants in Chandigarh and found that the male: female ratio was 2.7:1.[55]

SC Sharma et al also found that the male: female ratio was 2.6:1.[56]

SK Sayal et al conducted a study in Pune and found that the male: female ratio was 2.2:1.[57]

In a study by Suraj V Davis et al the male to female ratio was 2.05:1.[58]

The male: female ratio in a study conducted by Agarwal et al was 2.6:1.[59]

2. AGE:

In this study majority of the patients (39 %) were between the age group 30 – 40 years. The youngest was a 13 year old female and the oldest was 70 year old male. The mean age was 43.3 years.

SC Sharma et al in their study found that most of the patients (68%) were between the age group 20-40 years and the mean age was 40.1.[56]

In a study conducted by SK Sayal et al in Pune 64% of the patients belonged to the age group 20-49 years.[57]

Suraj V Davis et al in their study found that 44% belonged to the age group 30-50 years.[58]

In a study conducted by K Vinod Sharma et al in Chandigarh majority of the patients belonged to the age group 30-59 years.[55]

VK Sharma et al found that most of the patients belonged to the age group 22 – 70 years.[56]

3. **OCCUPATION:**

Occupational exposure was found in 69% of the patients.

In males 88.23 % had occupational exposure whereas in case of females non occupational exposure was common (71.87 %).

69% of the patients were exposed to the compositae plants occupationally as farmers (46%), labourers (20%), flower vendors (3%).

31% were exposed non occupationally with housewives being commonly affected (20%).

In a study by SC Sharma et al 84 % had occupational exposure.[56]

In a study conducted by Suraj V Davis et al agricultural workers accounted for 48% and 26% were housewives which is similar to our study.[58]

In a study by Agarwal et al 90 % were farmers.[59]

In a study conducted by K Vinod Sharma et al in Chandigarh 20.7% were farmers.[55]

4. DURATION OF OCCUPATIONAL EXPOSURE:

In this study the duration of occupational exposure ranged from 6 months to 50 years and is commonly more than 10 years.

In a study by Suraj V Davis et al the duration of occupational exposure ranged from 1 year to 50 years.[58]

5. SYMPTOMS:

In this study all the patients complained of itching and 4% had oozing and 7% had burning sensation.

6. DURATION OF COMPLAINTS:

In this study the duration of complaints ranged from 2 weeks to 10 years.

Most of them had complaints for less than a year.

This study shows that there is no correlation between duration of occupational exposure and onset of disease.

In a study by SC Sharma et al the duration ranged from 2 months to 10 years which is similar to this study.[56]

In a study by Suraj V Davis et al the duration of complaints ranged from 4 months to 26 years.[57]

7. SEASONAL VARIATION:

In this study 36% had summer exacerbation and 7% complained of winter exacerbation whereas 57% showed no seasonal variation.

SC Sharma et al found that 51% had summer exacerbation.[56]

Agarwal et al in their study found that 74.5% had summer exacerbation.[59]

Hemantha Kumar Kar et al in their study found summer exacerbation in 50%, exacerbation during rainy season in 2.1%%, in both summer and rainy season in 7%, winter exacerbation in 2.1% and no seasonal variation in 35.2%.[60]

8. ASSOCIATED ATOPY:

In this study 16% of the patients were atopics.

Occupational exposure was common among the atopics (81.25%).

In a study by Suraj V Davis et al 12% were atopics.[58]

8. PHOTOSENSITIVITY:

In this study history of photosensitivity was present in about 31% of the patients.

In a study by Suraj V Davis et al photosensitivity history was present in 62% of patients.[58]

In a study conducted by K Vinod Sharma et al in Chandigarh, 9.51% had photosensitivity.

10. CLINICAL PATTERN:

In this study the most common clinical pattern was air borne contact dermatitis (44%) followed by widespread dermatitis (20%) and phytophotodermatitis (16%). Hand eczema was found in 9%, erythroderma in 7%, localized dermatitis in 3 % and atopic like pattern in 1%.

Parthenium dermatitis commonly presented as air borne contact dermatitis (48.35%).

The most common clinical pattern in patients with contact dermatitis due to chrysanthemum was hand eczema (75%). One patient with chrysanthemum dermatitis presented with widespread dermatitis.

The patient with contact dermatitis due to helianthus presented as phytophotodermatitis.

The patient with contact dermatitis due to xanthium presented as hand eczema.

All patients with contact dermatitis due to garlic presented as hand eczema.

Among the farmers, ABCD was the commonest pattern observed.

Hand eczema was common among housewives.

In a study by Suraj V Davis et al 45.5% presented with air borne contact dermatitis which is similar to this present study.[58]

In a study by Agarwal et al 46% had ABCD, 30% had mixed pattern, 14% presented with erythroderma and 10% had chronic actinic dermatitis.[59]

In a study conducted by SK Sayal et al in Pune 68.25% presented with ABCD, 20% had phytophotodermatitis, 10% presented with erythroderma and 1.25% had localized dermatitis.[57]

SC Sharma et al found that among 54 patients with Parthenium dermatitis 34 had ABCD, 11 had involvement of face and hand only, 5 had photodermatitis, 3 had widespread dermatitis and 1 had erythroderma. Among 18 patients with Chrysanthemum dermatitis 11 had

ABCD, 4 had involvement of hand and face only, 2 had photodermatitis and 1 presented with widespread dermatitis.[56]

11. PATCH TEST RESULTS:

In this study 89% tested patch test positive for Parthenium alone, 1% for both Parthenium and Xanthium, 1% for both Parthenium and Helianthus, 4% for Chrysanthemum and 5% for garlic.

In a study by Agarwal et al 90% tested patch test positive for Parthenium.[59]

In a study by SC Sharma et al Parthenium produced positive reaction in 51% and Chrysanthemum in 23%.[56]

In a study by Hemanta Kumar Kar et al among 36 patients with plant sensitivity, 28 showed sensitivity to Parthenium alone, 5 showed sensitivity to both Parthenium and Xanthium and 3 showed sensitivity to Parthenium, Xanthium and Chrysanthemum. Isolated Xanthium and Chrysanthemum sensitivity was not seen in any patient.[60]

12. SEVERITY GRADING OF PATCH TEST REACTION:

In this study 47% had 1+ reaction, 43% had 2+ reaction and 10% had 3+ reaction. None had irritant reaction.

None of the patients developed adverse reactions to patch testing.

In a study by Abdur Rahim Khan et al, 14.8% showed 1+ reaction, 62.96% showed 2+ reaction and 22.2% had 3+ reaction.[61] None had irritant reaction

CONCLUSION

1. In this study among patients with phytodermatitis, males outnumbered females with a ratio of 2.1:1.

2. Most cases of allergic contact dermatitis to plants belonged to the age group 30 – 40 years.

3. Occupational exposure is more common than non occupational exposure. Farmers predominated the occupational group. In males occupational exposure is common whereas in females non occupational exposure is common.

4. The duration of occupational exposure ranges from 6 months to 50 years.

5. The duration of complaints ranged from 2 weeks to 10 years.

6. About half of them did not have any seasonal variation.

7. About 12% of the patients were atopics.

8. Air borne contact dermatitis was the most common pattern observed.

9. Parthenium was the most common offending allergen.

The drawbacks of this study:

1. Patch testing to common allergens only was done.
2. Photo patch testing was not done.

This study shows that Parthenium is still the commonest cause of phytodermatitis in our country, causing much distress and morbidity to the patients. This multi-faceted problem needs the cooperative endeavour of our botanists, biochemists, immunologists and dermatologists. Adequate measures must be taken to eradicate this notorious weed from our country.

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PROFORMA

- NAME: OP NO:
- AGE: OCD NO:
- SEX: ADDRESS:

OCCUPATION:

HISTORY OF PRESENT ILLNESS

- Chief complaints

  Onset

  Progression

  Exacerbating factors

  Seasonal variation

- H/O contact with allergen
- H/O photosensitivity
- H/O atopy

- PAST HISTORY :

  Similar illness in the past

  diabetes, hypertension

- TREATMENT H/O
- GENERAL EXAMINATION
- VITALS
- SYSTEMIC EXAMINATION

  CVS

  RS

  PER ABDOMEN

  CNS
- DERMATOLOGICAL EXAMINATION

  -MORPHOLOGY

  -SITE
- INVESTIGATIONS

  - Patch test
- DIAGNOSIS
- TREATMENT

S.No.	SEX	AGE	OCC	OCC. DUR	COMPLAINTS	DOC	SV	ATOPY	PS	SITES	MORPHOLOGY	PATTERN	AS.ALL	PATCH TEST	RESULTS
1	F	3	4	4	1,2	2	3	-	-	fingertips	sca, fissures	4	-	chrys	1+
2	M	1	5	-	1,2,3	1	3	-	-	ne, ch,cf	ery,sca	7	-	par	2+
3	F	3	3	-	1,3	5	3	fam h/o +	-	fingertips	ery,pap,ves	4	-	garlic	1+
4	F	4	3	-	1,2	2	3	-	-	palms,fingertips	pap, sca	4	-	garlic	1+
5	F	4	1	5	1	3	1	-	+	UL, Abd, ne	pap, pla, sca	2	-	par	2+
6	M	1	5	-	1	3	3	-	-	fin,handdorsa	pla,sca,pig	4	-	par, xan	1+ , 1+
7	M	4	6	-	1	3	1	-	-	fa,ne,UL,LL,E,RA	pla ,pig, sca	1	-	par	2+
8	F	3	3	-	1	2	3	-	-	fa,ne,UL,LL,E,RA	lic,sca, pla	1	-	par	3+
9	F	5	7	-	1	2	2	_	-	ne,trunk,UL,LL	lic,sca,pap,pla	3	-	par	neg
10	M	3	6	-	1	2	3	-	+	fa,ne,kn, fl	lic,pap,pla	2	-	par	1+
11	M	5	1	7	1	5	3	-	-	fa,trunk,UL,LL	lic,sca,pla	5	-	par	1+
12	F	3	1	6	1	3	3	-	+	ne,abd	lic,sca,pla	2	-	par	2+
13	F	1	5	-	1	2	3	+	-	fa,ne,UL,LL,E,RA	pig,sca,pla	1	-	par	3+
14	M	2	8	-	1	4	1	-	+	fa,ne,UL,LL,E,RA	pig,sca,pla	1	-	par	2+
15	M	5	2	6	1	1	3	+	+	fa,UL	pig, pap	2	-	par	1+
16	M	3	2	7	1,4	1	3	-	+	fa,trunk,UL,LL	ery,sca	5	cem	par	1+
17	M	5	7	-	1	1	3	+	+	fa,ne	sca,pig	2	-	par	2+
18	F	3	1	4	1	3	1	-	-	ne,arm,FA,ft	lic,pla	2	-	par,hel	3+,2+
19	M	3	4	4	1,3	2	3	-	-	fingertips	sca,fissures	4	-	chrys	2+
20	M	5	2	6	1	4	2	-	+	fa,ne,UL,LL,E,RA	pig,pla	1	-	par	2+
21	M	3	2	6	1	3	1	+	+	fa,ne,UL,LL,E,RA	ery,sca,pig	1	-	par	2+
22	F	3	3	-	1	2	3	-	+	fa,ne,UL,LL,E,RA	lic,pla	1	-	par	1+
23	M	5	1	7	1	3	1	-	+	fa,UL,LL	lic, pig	1	-	par	2+
24	F	3	3	-	1	4	3	-	-	hand,ft	lic,pla	6	-	par	2+

25	M	4	1	7	1	3	1	-	-	fa,trunk,UL,LL	ery,sca	5	-	par	1+
26	M	3	2	4	1	4	2	-	-	FA,ft,back	pig,lic,pla	3	-	par	1+
27	M	5	1	7	1	4	1	-	+	fa,ne,UL,LL,E,RA	lic,pig,pla	1	-	par	2+
28	F	3	1	2	1	3	2	+	+	fa,trunk,UL,LL	ery,sca	5	-	par	2+
29	F	3	4	4	1	4	1	-	+	trunk,FA,hand	lic,pla	3	-	par	1+
30	M	5	1	7	1	4	2	-	+	fa,hands,ft	lic,pla	1	-	par	2+
31	M	3	1	4	1	3	3	-	+	both foot	lic,pla	6	-	par	1+
32	F	5	3	-	1,2	2	3	-	-	fingertips	sca,fissures	4	-	garlic	1+
33	F	2	3	-	1	2	3	-	-	fingertips	sca,fissures	4	-	garlic	2+
34	M	4	1	7	1	2	3	-	+	fa,ne,UL,LL,E,RA	lic,pig,pla	1	-	par	2+
35	F	3	1	4	1	2	3	_	_	fingertips	sca,fissures	4	-	chrys	1+
36	M	4	1	7	1	3	2	-	-	fa,ne,UL	lic,pla,pig	3	-	par	1+
37	M	5	2	7	1	3	2	-	-	fa,ne,trunk,UL	lic,pla,pig	3	-	par	1+
38	M	3	2	6	1	4	1	-	-	fa,ne,UL	lic,pla,pig	2	-	par	2+
39	F	5	3	-	1	3	1	-	-	both UL	lic,pla,sca	3	-	par	1+
40	F	1	5	-	1	2	3	-	-	fa,trunk,UL,LL	lic,sca	3		par	1 +
41	M	2	2	4	1	2	3	-	-	fa,UL	lic,pla,sca	3	cem	par,p,n,c	1+,1+,neg,neg
42	M	3	2	6	1	3	2	+	-	fa,ne,UL,LL,E,RA	lic,pla,sca	1	cem	par,p,n,c	1+,1+,neg,neg
43	M	2	1	3	1	1	3	-	-	fa,ne,UL	lic,pla,sca	2	-	par	1+
44	F	3	1	4	1	2	3	-	-	fa,ne,UL	lic,pla,sca	2	-	par	3+
45	M	4	6	-	1	3	1	-	+	fa,ne,UL	lic,pla,sca	2	-	par	2+
46	F	4	3	-	1	1	3	-	-	fa,ne,UL,LL	lic,pla,sca	3	-	par	1+
47	M	5	2	7	1	2	3	-	-	fa,ne,UL	lic,pla,sca	2	-	par	2+
48	M	4	1	4	1	3	2	-	-	fa,ne,trunk,UL	lic,pla,sca	3	-	par	2+
49	M	4	1	5	1	3	1	-	-	fa,ne,UL	lic,sca,pla	2	-	par	2+
50	M	4	6	-	1	2	3	-	-	fa,ne,UL,LL,E,RA	lic,pla,sca	1	-	par	1+

51	F	3	3	-	1	1	3	-	-	fa,ne	lic,sca,pla	2	cem	par,ni,co	1+,1+,1+
52	F	5	3	-	1	2	3	-	-	fa,ne,trunk,UL	lic,sca,pla	3	-	par	1+
53	M	3	1	4	1	2	3	-	-	fa,ne,trunk,UL	lic,sca,pla	3	-	par	2+
54	F	5	1	7	1	2	3	+	+	fa,trunk,UL,LL	lic,sca,pla	5	-	par	3+
55	M	4	1	4	1	2	3	-	-	fa,ul	lic,sca,pla	3	cem	par,chrys,ni	2+,2+,1+
56	M	5	2	8	1	3	1	+	+	fa,ne,UL	lic,sca,pla	2	cem	par, ni	2+,1+
57	M	4	1	4	1	1	3	-	-	fa,ne,trunk,UL	lic,sca,pla	3	-	par	1+
58	M	3	2	4	1	2	3	-	-	fa,ne,trunk,UL	lic,sca,pla	3	-	par	1+
59	M	3	1	3	1	3	1	-	-	fa,ne	lic,sca,pla	3	-	par	2+
60	F	5	3	-	1	2	3	-	-	fa,ne,UL,LL	lic pla,sca	1	-	par	1+
61	M	5	2	8	1	1	3	-	-	fa,ne,UL,LL,E,RA	lic pla,sca	1	-	par	1+
62	M	5	1	6	1	3	1	-	+	fa,trunk,UL,LL	ery,sca	5	-	par	2+
63	M	5	2	8	1	3	1	-	-	fa,ne,UL,LL,E,RA	lic pla,sca	1	-	par	3+
64	M	5	1	8	1	1	3	+	-	fa,ne,UL,LL,E,RA	lic pla,sca	1	-	par	3+
65	M	3	2	4	1	3	1	-	+	fa,ne,UL,LL,E,RA	lic pla,sca	1	-	par	3+
66	M	5	1	8	1	3	2	-	-	fa,ne,trunk,UL	lic pla,sca	3	-	par	3+
67	M	5	1	5	1	1	3	+	-	fa,ne,UL,LL,E,RA	lic pla,sca	1	-	par	3+
68	M	5	1	8	1	2	3	-	+	fa,ne,UL,LL,E,RA	lic pla,sca	1	-	par	2+
69	F	3	3	-	1	2	3	-	-	feet	lic pla,sca	6	-	par	2+
70	M	3	1	4	1	1	3	-	+	fa,ne,UL,LL,E,RA	lic pla,sca	1	-	par	2+
71	F	5	3	-	1	1	3	-	-	fa,ne,UL,LL,E,RA	lic pla,sca	1	-	par	1+
72	M	5	2	5	1	4	1	-	+	fa,ne,UL,LL,E,RA	lic pla,sca	1	cem	par,ni	2+,1+
73	M	4	1	6	1	1	3	+	-	fa,ne,UL,LL,E,RA	lic pla,sca	1	-	par	1+
74	M	5	1	8	1	2	3	-	-	fa,ne,UL,LL,E,RA	lic pla,sca	1	-	par	1+
75	F	3	3	-	1	1	3	-	-	fa,ne,UL,LL,E,RA	lic pla,sca	1	-	par	2+
76	M	3	1	4	1	1	3	-	-	fa,ne,UL,LL,E,RA	lic pla,sca	1	-	par	1+

77	F	4	3	-	1	1	3	-	-	hands	sca,fissures	4	-	garlic	1+
78	M	4	1	6	1	3	1	-	+	fa,ne trunk,UL	lic pla,sca	3	-	par	1+
79	M	5	2	8	1	1	3	-	-	fa,ne,UL,LL,E,RA	lic pla,sca	1	-	par	1+
80	M	4	1	6	1	3	1	-	+	fa,ne,UL,LL,E,RA	lic pla,sca	1	-	par	1+
81	M	5	1	8	1	1	3	+	-	fa,ne,UL,LL,E,RA	lic pla,sca	1	-	par	2+
82	M	3	2	5	1	3	2	-	+	fa,ne UL	lic pla,sca	2	-	par	1+
83	F	5	3	-	1	3	3	-	-	fa,trunk,UL,LL	ery,sca	5	-	par	1+
84	F	4	3	-	1	1	3	-	-	fa,ne,UL,LL,E,RA	lic pla,sca	1	-	par	1+
85	F	2	3	-	1	3	1	-	+	fa,ne,UL,LL,E,RA	lic pla,sca	1	-	pa	1+
86	F	5	3	-	1	3	1	-	-	fa,ne,UL,LL,E,RA	lic pla,sca	1	-	par	2+
87	M	3	1	6	1	1	3	-	-	fa,ne,UL,LL,E,RA	lic pla,sca	1	-	par	2+
88	M	4	1	8	1	5	1	-	-	fa,ne,trunk,UL	lic pla,sca	3	-	par	1+
89	M	3	2	4	1	3	2	-	+	fa,ne,UL,LL,E,RA	lic pla,sca	1	-	par	2+
90	M	4	1	8	1	1	3	+	-	fa,ne,UL,LL,E,RA	lic pla,sca	1	-	par	2+
91	M	3	1	6	1	1	3	-	-	fa,ne,UL,LL,E,RA	lic pla,sca	1	-	par	1+
92	M	3	1	5	1	4	1	-	-	fa,ne,UL,LL,E,RA	lic pla,sca	1	-	par	2+
93	M	4	1	8	1	3	2	-	-	fa,ne,UL,LL,E,RA	lic pla,sca	1	-	par	1+
94	M	3	2	5	1	1	3	-	+	fa,UL,	lic pla,sca	2	-	par	1+
95	M	3	1	6	1	3	1	-	-	fa,ne,UL,LL,E,RA	lic pla,sca	1	-	par	2+
96	M	3	1	5	1	3	1	+	-	fa,ne,UL,LL,E,RA	lic pla,sca	1	-	par	2+
97	M	4	2	5	1	4	1	-	-	fa,ne,UL,LL,E,RA	lic pla,sca	1	-	par	1+
98	M	3	1	6	1	1	3	-	-	fa,ne,UL,LL,E,RA	lic pla,sca	1	-	par	2+
99	M	3	1	5	1	4	1	-	-	fa,ne,UL,LL,E,RA	lic pla,sca	1	-	par	2+
100	M	3	2	5	1	3	1	+	-	fa,ne,trunk,UL	lic pla,sca	3	-	par	2+

KEY TO MASTER CHART

SEX

M – Male

F – Female

AGE

1 = 0-10 years

2 = 10-20 years

3 = 20-30 years

4 = 30-40 years

5 = 40-50 years

5 = 10-20 years

6 = 20-30 years

7 = 30-40 years

8 = 40-50 years

OCCUPATION (occ)

1 – Farmer

2 - Labourer

3 - Housewife

4 - Flower vendor

5 - Student

6 – Watchman

7 – Weaver

8 - Driver

COMPLAINTS(COMP)

1 - itching

2 – scaling

3 – oozing

4 - burning

DURATION OF COMPLAINTS(DOC)

1 = <1 month

2 = 1-6 months

3 = 6 month-1 year

4 = 1-5 years

5 = 5-10 years

SEASONAL VARIATION(SV)

1 = summer exacerbation

2 = winter exacerbation

3 = no seasonal variation

ATOPY

+ → present

- → absent

PHOTOSENSITIVITY(PS)

+ → present

- → absent

SITES

ne - neck

ch - chest

cf - cubital fossa

UL - upper limb

abd - abdomen

fa - face

LL - lower limb

Kn - knee

Fl - flexures

fa - forearm

ft - feet

MORPHOLOGY

Sca - scaling

Ery - erythema

Pap - papule

Ves - vesicle

Pla - plaque

Pig - pigmentation

Lic - lichenification

PATTERN

1 - Air Borne Contact Dermatitis

2 - Phytophotodermatitis

3 - Widespread dermatitis

4 - Hand eczema

5 - Erythroderma

6 - Localized

7 - Atopic like

ALLERGENS

Par - Parthenium

Chrys - chrysanthemum

Hel - helianthus

Xan - xanthium

p - potassium di chromate

ni - nickel

co - cobalt

Cem - Cement

AS.ALL - Associated Allergens

ABBREVIATIONS

NSAIDS	–	Non steroidal anti inflammatory drugs.
UVR	–	Ultra violet rays.
TNF	–	Tumour Necrosis Factor
GM-CSF	–	Granulocyte Monocyte-Colony Stimulating Factor.
IL	–	Interleukin.
ABCD	–	Air Borne Contact Dermatitis.
CAD	–	Chronic Actinic Dermatitis.
SQL	–	Sesquiterpene lactones

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI -3

Telephone No : 044 25305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr. S. Sasirekha
PG in MDDVL
Madras Medical College, Chennai -3

Dear Dr. S. Sasirekha

The Institutional Ethics committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled "Clinicoepidemiologic study of phytodermatitis" No. 04122011

The following members of Ethics Committee were present in the meeting held on 22.12.2011 conducted at Madras Medical College, Chennai -3.

1. Prof. S.K. Rajan. MD -- Chairperson
2. Prof. R. Nandhini MD -- Member
   Director, Institute of Pharmacology ,MMC, Ch-3
3. Prof. Pregna B. Dolia MD -- Member
   Director , Institute of Biochemistry, MMC, Ch-3
4. Prof. S. Regunanthan, MD -- Member
   Prof of Internal Medicine, MMC, Ch-3
5. Prof. Md Ali MD. DM -- Member
   Prof & Head , Dept. of MGE, MMC,Ch-3
6. Thiru. S. Govindsamy. BA BL -- Lawyer

We approve the proposal to be conducted in its presented form.

Sd/ Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

15-1-

Member Secretary, Ethics Committee